



Blast 2 Sequences results

PubMed

Entrez

BLAST

OMIM

Taxonomy

Structure

BLAST 2 SEQUENCES RESULTS VERSION BLASTN 2.2.2 [Dec-14-2001]

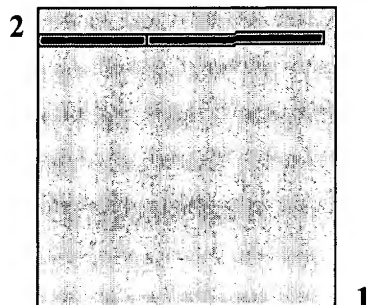
Match: Mismatch: gap open: gap extension:
 x_dropoff: expect: wordsize: Filter ☒ Align

Sequence 1 lcl|seq_1

Length 22 (1 .. 22)

Sequence 2 gi 3006202 Homo sapiens stanniocalcin precursor (STC) mRNA, complete cds.

Length 3901 (1 .. 3901)



NOTE: The statistics (bitscore and expect value) is calculated based on the size of nr database

NOTE: If protein translation is reversed, please repeat the search with reverse strand of the query sequence

Score = 29.5 bits (15), Expect = 5.6
 Identities = 22/23 (95%), Gaps = 1/23 (4%)
 Strand = Plus / Plus

Query: 1 cctccaatt-cccccttaaactt 22
 ||||| |||||
 Sbjct: 3465 cctccaattcccccttaaactt 3487

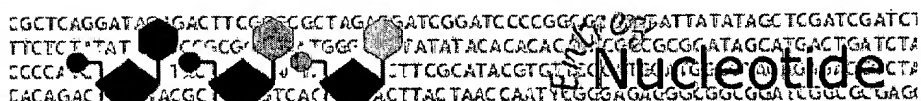
CPU time: 0.02 user secs. 0.04 sys. secs 0.06 total secs.

Gapped
 Lambda K H
 1.33 0.621 1.12

Gapped
 Lambda K H
 1.33 0.621 1.12

Matrix: blastn matrix:1 -2
 Gap Penalties: Existence: 5, Extension: 2
 Number of Hits to DB: 3
 Number of Sequences: 0
 Number of extensions: 3
 Number of successful extensions: 3
 Number of sequences better than 10.0: 1

length of query: 22
length of database: 4,611,916,737
effective HSP length: 21
effective length of query: 1
effective length of database: 4,309,374,105
effective search space: 4309374105
effective search space used: 4309374105
T: 0
A: 30
X1: 6 (11.5 bits)
X2: 26 (50.0 bits)
S1: 12 (23.8 bits)
S2: 15 (29.5 bits)



1: U46768. Human stanniocalcin 2 (STC2) mRNA, partial cDNA sequence [Related Sequences, OMIM, Protein, Taxonomy, LinkOut](#)

1	aaaacccaac	aacttagcgg	aaacttctca	gagaatgctc	caaaactcag	cagtgtctct
61	ggtgctggtg	atcagtgtct	ctgcaaccca	tgaggcggag	cagaatgact	ctgtgagccc
121	caggaaatcc	cgagtggcgg	cccaaaactc	agctgaagtg	gttcgttgcc	tcaacagtgc
181	tctacaggtc	ggctgcgggg	cttttgcatg	cctggaaaac	tccacctgtg	acacagatgg
241	gatgtatgac	atctgtaaat	ccttcttgta	cagcgtgtgt	aaatttgaca	ctcagggaaa
301	agcattcgct	aaagagagct	taaaattgcat	cgccaactgg	gtcacctcca	aggctctctc
361	cgccattcgg	agggtgtcca	ctttccaaag	gatgattggg	gaggtgcagg	aagagtgtcta
421	cagcaagctg	aatgtgtgca	gcatcgccaa	gcggaaccct	gaagccatca	ctgaggtcgt
481	ccagctgccc	aatcacttct	ccaacagata	ctataacaga	cttgtccgaa	gcctgtctgga
541	atgtgatgaa	gacacagtca	gcacaatcag	agacagcctg	atggagaaaa	ttgggcctaa
601	catggccagc	ctcttccaca	tcctgcagac	agaccactgt	gccc aaacac	acccacgagc
661	tgacttcaac	aggagacgca	ccaatgagcc	gcagaagctg	aaagtctctc	tcaggaaactc
721	ccggagttag	gaggactctc	cctcccatat	caaaagcaca	tcccatgaga	gtgcataacc
781	agggagaggt	tattcacaaac	ctcaccaaac	tagtatcatt	ttaggggtgt	tgacacacc

Revised: October 24, 2001.

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[NCBI](#) | [NLM](#) | [NIH](#)

>gi|16162450|ref|XM_011704.4| Homo sapiens stanniocalcin 1 (STC1), mRNA
Length = 3877

Score = 40.1 bits (20), Expect = 0.011
Identities = 20/20 (100%)
Strand = Plus / Minus

Query: 1 tctaggtcagcccccgaaac 20
|||||||
Sbjct: 3536 tctaggtcagcccccgaaac 3517

>gi|4507264|ref|NM_003155.1| Homo sapiens stanniocalcin 1 (STC1), mRNA
Length = 3901

Score = 40.1 bits (20), Expect = 0.011
Identities = 20/20 (100%)
Strand = Plus / Minus

Query: 1 tctaggtcagcccccgaaac 20
|||||||
Sbjct: 3538 tctaggtcagcccccgaaac 3519

>gi|6716576|gb|AF178116.1|AF178116 Canis familiaris STC1 gene, complete sequence
Length = 1053

Score = 40.1 bits (20), Expect = 0.011
Identities = 20/20 (100%)
Strand = Plus / Plus

Query: 1 tctaggtcagcccccgaaac 20
|||||||
Sbjct: 343 tctaggtcagcccccgaaac 362

>gi|3006202|gb|U25997.1|HSU25997 Homo sapiens stanniocalcin precursor (STC) mRNA,
complete cds
Length = 3901

Score = 40.1 bits (20), Expect = 0.011
Identities = 20/20 (100%)
Strand = Plus / Minus

Query: 1 tctaggtcagcccccgaaac 20
|||||||
Sbjct: 3538 tctaggtcagcccccgaaac 3519

>gi|261373|gb|S50179.1|S50179 {clone S1} [human, 76N, mammary epithelial cells, mRNA,
607 nt]

Length = 607

Score = 40.1 bits (20), Expect = 0.011

Identities = 20/20 (100%)

Strand = Plus / Minus

Query: 1 tctagggtcagccccgaatc 20

||||||||||||||||

Sbjct: 244 tctagggtcagccccgaatc 225

**NCBI**
Nucleotide Protein Translations Retrieve results for an RID

formatting **BLAST**

Your request has been successfully submitted and put into the Blast Queue.

Query = (20 letters)

The request ID is

Format! or **Reset all**

The results are estimated to be ready in 2 minutes 10 seconds but may be done sooner.

Please press "FORMAT!" when you wish to check your results. You may change the formatting options for your result via the form below and press "FORMAT!" again. You may also request results of a different search by entering any other valid request ID to see other recent jobs.

Format

Show ☒ Graphical Overview ☒ NCBI-gi in format

Number of: Descriptions

Alignment view

Limit results by or select from:

Expect value
range:

 **NCBI**
Nucleotide Protein Translations Retrieve results for an RID

formatting **BLAST**

Your request has been successfully submitted and put into the Blast Queue.

Query = (22 letters)

The request ID is

Format! or **Reset all**

The results are estimated to be ready in 2 minutes 10 seconds but may be done sooner.

Please press "FORMAT!" when you wish to check your results. You may change the formatting options for your result via the form below and press "FORMAT!" again. You may also request results of a different search by entering any other valid request ID to see other recent jobs.

Format

Show ☒ Graphical Overview ☒ NCBI-gi Alignment in format

Number of: Descriptions Alignments

Alignment view

Limit results by or select from:

Expect value
range:



Blast 2 Sequences results

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BLAST 2 SEQUENCES RESULTS VERSION BLASTN 2.2.2 [Dec-14-2001]

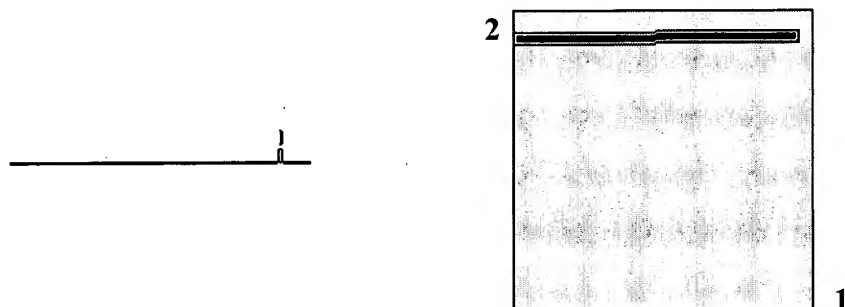
Match: Mismatch: gap open: gap extension:
 x_dropoff: expect: wordsize: Filter ☒ Align

Sequence 1 lcl|seq_1

Length 20 (1 .. 20)

Sequence 2 gi 3006202 Homo sapiens stanniocalcin precursor (STC) mRNA, complete cds.

Length 3901 (1 .. 3901)



NOTE: The statistics (bitscore and expect value) is calculated based on the size of nr database

NOTE: If protein translation is reversed, please repeat the search with reverse strand of the query sequence

Score = 39.1 bits (20), Expect = 0.007
 Identities = 20/20 (100%)
 Strand = Plus / Plus

Query: 1 gattcgggggctgacctaga 20
 |||||
 Sbjct: 3519 gattcgggggctgacctaga 3538

CPU time: 0.03 user secs. 0.05 sys. secs 0.08 total secs.

Gapped
 Lambda K H
 1.33 0.621 1.12

Gapped
 Lambda K H
 1.33 0.621 1.12

Matrix: blastn matrix:1 -2
 Gap Penalties: Existence: 5, Extension: 2
 Number of Hits to DB: 1
 Number of Sequences: 0
 Number of extensions: 1
 Number of successful extensions: 1
 Number of sequences better than 10.0: 1

length of query: 20
length of database: 4,611,916,737
effective HSP length: 19
effective length of query: 1
effective length of database: 4,310,814,769
effective search space: 4310814769
effective search space used: 4310814769
T: 0
A: 30
X1: 6 (11.5 bits)
X2: 26 (50.0 bits)
S1: 12 (23.8 bits)
S2: 15 (29.5 bits)

Query: 501 ttaaagatgccatagcataatatgaagcctttggtgaattccttctaagataaaaaataat 560
|||||
Sbjct: 501 ttaaagatgccatagcataatatgaagcctttggtgaattccttctaagataaaaaataat 560

Query: 561 aataaagtgttacgttttatttggtttc 587
|||||
Sbjct: 561 aataaagtgttacgttttatttggtttc 587

>gi|4507264|ref|NM_003155.1| Homo sapiens stanniocalcin 1 (STC1), mRNA
Length = 3901

Score = 1067 bits (538), Expect = 0.0
Identities = 559/567 (98%), Gaps = 1/567 (0%)
Strand = Plus / Plus

Query: 21 ggctgtgacctcttcaaaccgtggtannnnnnnttttctccccacgatgatatctatata 80
|||||
Sbjct: 3316 ggctgtgacctcttcaaaccgtgtaccccccttttctccccacgatgatatctatata 3375

Query: 81 tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatggtgctag 140
|||||
Sbjct: 3376 tgtatctacaatacatatatctacacatacagaaagaagcagttctcaca-tggtgctag 3434

Query: 141 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 200
|||||
Sbjct: 3435 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 3494

Query: 201 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcattgattc 260
|||||
Sbjct: 3495 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcattgattc 3554

Query: 261 ttctcttggtgatttggttgcaacttttagacatttttgtgccattatatttgcaattatgtat 320
|||||
Sbjct: 3555 ttctcttggtgatttggttgcaacttttagacatttttgtgccattatatttgcaattatgtat 3614

Query: 321 ttataatttaaatagatatttaggtttttggctgagtactggaataaacagtgagcatatc 380
|||||
Sbjct: 3615 ttataatttaaatagatatttaggtttttggctgagtactggaataaacagtgagcatatc 3674

Query: 381 tggatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 440
|||||
Sbjct: 3675 tggatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 3734

Alignments

>gi|261373|gb|S50179.1|S50179 {clone S1} [human, 76N, mammary epithelial cells, mRNA, 607 nt]

Length = 607

Score = 1082 bits (546), Expect = 0.0

Identities = 560/567 (98%)

Strand = Plus / Plus

```

Query: 21  ggctgtgacctcttcaaaccgtggtannnnnnnttttctccccacgatgatatctatata 80
          |||
Sbjct: 21  ggctgtgacctcttcaaaccgtggtaccccccttttctccccacgatgatatctatata 80

Query: 81  tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatgttgctag 140
          |||
Sbjct: 81  tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatgttgctag 140

Query: 141 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 200
          |||
Sbjct: 141 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 200

Query: 201 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 260
          |||
Sbjct: 201 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 260

Query: 261 ttctcttggtgatttggttgcaacttttagacatttttggtgccattatatttgcatatgtat 320
          |||
Sbjct: 261 ttctcttggtgatttggttgcaacttttagacatttttggtgccattatatttgcatatgtat 320

Query: 321 ttataatttaaatagatatttaggttttggtgagtgactggaataaacagtgagcatatc 380
          |||
Sbjct: 321 ttataatttaaatagatatttaggttttggtgagtgactggaataaacagtgagcatatc 380

Query: 381 tggatatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 440
          |||
Sbjct: 381 tggatatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 440

Query: 441 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 500
          |||
Sbjct: 441 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 500

```

Query: 441 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 500
 |||
 Sbjct: 3735 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 3794

Query: 501 tttaaagatgccatagcataatatgaagcctttgggtgaattccttctaagataaaaaataat 560
 |||
 Sbjct: 3795 tttaaagatgccatagcataatatgaagcctttgggtgaattccttctaagataaaaaataat 3854

Query: 561 aataaagtgttacgttttatttggtttc 587
 |||
 Sbjct: 3855 aataaagtgttacgttttatttggtttc 3881

>gi|3006202|gb|U25997.1|HSU25997 Homo sapiens stanniocalcin precursor (STC) mRNA,
 complete cds

Length = 3901

Score = 1067 bits (538), Expect = 0.0
 Identities = 559/567 (98%), Gaps = 1/567 (0%)
 Strand = Plus / Plus

Query: 21 ggctgtgacctcttcaaaccgtggtannnnnnnttttctccccacgatgatattctatata 80
 |||
 Sbjct: 3316 ggctgtgacctcttcaaaccgtggtaccccccttttctccccacgatgatattctatata 3375

Query: 81 tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatgttgctag 140
 |||
 Sbjct: 3376 tgtatctacaatacatatatctacacatacagaaagaagcagttctcaca-tgttgctag 3434

Query: 141 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 200
 |||
 Sbjct: 3435 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 3494

Query: 201 ttcgtcttggtgtttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 260
 |||
 Sbjct: 3495 ttcgtcttggtgtttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 3554

Query: 261 ttctcttgatgttggttgcaacttttagacatttttggtgccattatatttgcaattatgtat 320
 |||
 Sbjct: 3555 ttctcttgatgttggttgcaacttttagacatttttggtgccattatatttgcaattatgtat 3614

Query: 321 ttataatttaaatgatatttaggttttggctgagtagtactggaataaacagtgagcatatc 380

|||||
Sbjct: 3615 ttataatttaaagatatttaggttttggctgagtactggaataaacagtgagcatatc 3674

Query: 381 tggatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 440
|||||
Sbjct: 3675 tggatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 3734

Query: 441 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 500
|||||
Sbjct: 3735 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 3794

Query: 501 ttaaagatgccatagcataaatatgaagcctttggtgaattccttctaagataaaaaataat 560
|||||
Sbjct: 3795 ttaaagatgccatagcataaatatgaagcctttggtgaattccttctaagataaaaaataat 3854

Query: 561 aataaagtgttacgttttatttggtttc 587
|||||
Sbjct: 3855 aataaagtgttacgttttatttggtttc 3881

>gi|16162450|ref|XM_011704.4| Homo sapiens stanniocalcin 1 (STC1), mRNA
Length = 3877

Score = 1035 bits (522), Expect = 0.0
Identities = 557/567 (98%), Gaps = 3/567 (0%)
Strand = Plus / Plus

Query: 21 ggctgtgacctcttcaaaccgtggtannnnnnnttttctccccacgatgatatctatata 80
|||||
Sbjct: 3314 ggctgtgacctcttcaaaccgtggtaccccccttttctccccacgatgatatctatata 3373

Query: 81 tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatggtgctag 140
|||||
Sbjct: 3374 tgtatctacaatacatatatctacacatacagaaagaagcagttctcacaatggtgctag 3433

Query: 141 ttttttgcttctctttccccaccctactccctccaattcccccttaaacttccaaagc 200
|||||
Sbjct: 3434 ttttttgcttctctttccccaccctactccctccaattcccc-ttaaacttccaaagc 3492

Query: 201 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 260
|||||
Sbjct: 3493 ttcgtcttggtgttgctgcagagtgattcgggggctgacctagaccagtttgcatgattc 3552

Query: 261 ttctcttgatgatttggttgcaacttttagacatttttggtgccattatatttgcatatgtat 320
|||||
Sbjct: 3553 ttctcttgatgatttggttgcaacttttagacatttttggtgccattatatttgcatatgtat 3612

Query: 321 ttataatttaaatagatatttaggttttggtgagtgactggaataaacagtgagcatatc 380
|||||
Sbjct: 3613 ttataatttaaatagatatttaggttttggtgagtgactggaataaacagtgagcatatc 3672

Query: 381 tggatatgtcattatttattgttaaattacattttttaagctccatgtgcatataaagg 440
|||||
Sbjct: 3673 tggatatgtcattatttattgttaaattaca-tttttaagctccatgtgcatataaagg 3731

Query: 441 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgcttattttttataa 500
|||||
Sbjct: 3732 ttatgaaacatatcatggtaatgacagatgcaagttattttatttgctta-tttttataa 3790

Query: 501 ttaaagatgcatagcataaatatgaagcctttggtgaattccttctaagataaaaaataat 560
|||||
Sbjct: 3791 ttaaagatgcatagcataaatatgaagcctttggtgaattccttctaagataaaaaataat 3850

Query: 561 aataaagtgttacgttttatttggtttc 587
|||||
Sbjct: 3851 aataaagtgttacgttttatttggtttc 3877

(FILE 'HOME' ENTERED AT 15:45:20 ON 20 JAN 2002)

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS' ENTERED AT
15:45:52

ON 20 JAN 2002

L1 703 S STANNIOCALCIN OR STANNIUS
L2 35 S L1 AND (MESOTHELIOMA# OR CANCER# OR TUMOR# OR TUMOUR# OR
CARC
L3 18 S L2 AND PY<2000
L4 16 S L1 AND (MALIGNAN? OR METASTA? OR NEOPLAS?)
L5 5 S L4 AND PY<2000
L6 18 S L3 OR L5
L7 9 DUP REM L6 (9 DUPLICATES REMOVED)

FILE 'REGISTRY' ENTERED AT 16:03:34 ON 20 JAN 2002

L8 44 S STANNIOCALCIN

FILE 'CAPLUS' ENTERED AT 16:10:06 ON 20 JAN 2002

L9 139 S L8
L10 19 S L9 AND (TUMOR# OR TUMOUR# OR CANCER# OR MALIGNAN? OR NEOPLAS

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS' ENTERED AT
16:31:03

ON 20 JAN 2002

L11 0 S L1 AND (LEUKEMIA# OR LEUKAEMIA# OR LEUKEAMIA# OR LYMPHOMA#
OR

FILE 'JAPIO' ENTERED AT 16:47:13 ON 20 JAN 2002

L12 5 S STANNIOCALCIN

FILE 'SCISEARCH' ENTERED AT 16:52:08 ON 20 JAN 2002

L13 1762 S REDDEL?/RAU
L14 0 S L13(S)3/RVL(S)299/RPG
L15 4 S L13(S)1996/RPY

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS' ENTERED AT
16:56:47

ON 20 JAN 2002

L16 20 S L1 AND REDDEL?/AU
L17 20 S L1 AND CHANG?/AU
L18 20 S L16 OR L17
L19 15 S L18 AND PY<2000
L20 6 DUP REM L19 (9 DUPLICATES REMOVED)
L21 25 S LNG108 OR LUNG108 OR (LNG(A)108) OR (LUNG(A)108)
L22 11 DUP REM L21 (14 DUPLICATES REMOVED)

=>

<u>DB Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
JPAB,EPAB,DWPI	113 and (leukemia\$1 or leukaemia\$1 or leukeamia\$1 or lymphoma\$1 or myeloma\$1)	2	<u>L14</u>
JPAB,EPAB,DWPI	stanniocalcin or stannius	23	<u>L13</u>
USPT	110 and @prad<19991104	0	<u>L12</u>
USPT	110 and @ad<19991104	3	<u>L11</u>
USPT	19 and (leukemia\$1 or leukaemia\$1 or leukeamia\$1 or lymphoma\$1 or myeloma\$1)	4	<u>L10</u>
USPT	stanniocalcin or stannius	8	<u>L9</u>
USPT	17 or 16	37	<u>L8</u>
USPT	15 and @prad<19991104	2	<u>L7</u>
USPT	15 and @ad<19991104	37	<u>L6</u>
USPT	13 not 14	38	<u>L5</u>
USPT	12 same (leukemia\$1 or leukaemia\$1 or leukeamia\$1 or lymphoma\$1 or myeloma\$1)	3	<u>L4</u>
USPT	12 and (leukemia\$1 or leukaemia\$1 or leukeamia\$1 or lymphoma\$1 or myeloma\$1)	41	<u>L3</u>
USPT	stc\$1 or stanniocalcin or stannius	2799	<u>L2</u>
USPT	stc\$1 or stanniocalcin or stannius	2799	<u>L1</u>

<u>DB Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
USPT	112 and 1426	1	<u>L19</u>
USPT	112 and (SEQ ID NO: 1426)	0	<u>L18</u>
USPT	112 and (SEQ ID NO:1426)	0	<u>L17</u>
USPT	114 and 111	0	<u>L16</u>
USPT	114 and 110	0	<u>L15</u>
USPT	prostate cancer antigen\$1	10	<u>L14</u>
USPT	110 and 111	5	<u>L13</u>
USPT	110 or 111	78	<u>L12</u>
USPT	18 and ruben\$10[in]	15	<u>L11</u>
USPT	18 and rosen\$10[in]	68	<u>L10</u>
USPT	18 and rosen\$[in]	68	<u>L9</u>
USPT	prostate	8488	<u>L8</u>
USPT	rosen\$[in]	5313	<u>L7</u>
USPT	14 and @prad<19991104	0	<u>L6</u>
USPT	14 and @ad<19991104	7	<u>L5</u>
USPT	13 and (cancer\$1 or tumor\$1 or tumour\$1 or mesothelioma\$1 or carcinoma\$1 or adenocarcinoma\$1 or malignan\$4)	8	<u>L4</u>
USPT	11 or 12	8	<u>L3</u>
USPT	stanniocalcin	8	<u>L2</u>
USPT	stannius adj2 protein\$1	4	<u>L1</u>

L21 ANSWER 1 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 1999:476721 SCISEARCH
 THE GENUINE ARTICLE: 206MU
 TITLE: Preliminary studies on tin miners' lung **cancer**
 tissue related genes by differential display **mRNA**
 AUTHOR: Yang M Y (Reprint); Ye C Q; Yao S X; Zhang J; Chen J Y;
 Liu L H
 CORPORATE SOURCE: INST RADIAT MED, BEIJING 100850, PEOPLES R CHINA
 (Reprint); CHINA NATL NONFERROUS MET IND CORP, YUNNAN TIN
 CORP, INST LABOR PROTECT, GEJIU 661400, PEOPLES R CHINA
 COUNTRY OF AUTHOR: PEOPLES R CHINA
 SOURCE: CHINESE MEDICAL JOURNAL, (JUN 1999) Vol. 112,
 No. 6, pp. 529-533.
 Publisher: CHINESE MEDICAL ASSOCIATION, 42 DONGSI
 XIDAJIE,
 BEIJING 100710, PEOPLES R CHINA.
 ISSN: 0366-6999.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: CLIN
 LANGUAGE: English
 REFERENCE COUNT: 13

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective To study the genes related to tin miners' lung cancer
 tissue.

Method Differential display mRNA.

Results Thirty cDNA fragments which differentially expressed in lung
 cancer tissues and the same patient's normal lung tissues were
 discovered.

Among these, 16 expressed in lung cancer tissues, not in normal lung
 tissues; fourteen expressed on the contrary. Six cDNA fragment sequence
 was determined. Five sequences CG2, CG7, CG8, CA5 and CC6 had less than
 75% homology with known sequences in GenBank BLAST, so they were believed
 to be new sequences which we have recorded in Genbank. Only one fragment
 coded CG3 had homology up to 95% with human ribosome protein L27a gene.

Conclusions mRNA differential display provides a unique and powerful
 experimental system to study differential gene expression in tin miners'
 lung cancer tissues and the same patient's normal lung tissues. Using the
 system, differential expression of 30 cDNA fragments was observed. Six of
 them may be used to study the molecular mechanism of miners'
 radon-associated lung cancer.

L21 ANSWER 2 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 1999:310928 SCISEARCH
 THE GENUINE ARTICLE: 187GQ
 TITLE: Reduced expression of the CXCR4 receptor **mRNA** in
 hepatocellular **carcinoma** and lack of
 inducibility of its ligand alpha-chemokine hIRH/SDF1
 alpha/PBSF in vitro
 AUTHOR: Begum N A; Shibuta K; Mori M; Barnard G F (Reprint)
 CORPORATE SOURCE: UNIV MASSACHUSETTS, MED CTR, DIV DIGEST DIS & NUTR S6
 737,
 55 LAKE AVE N, WORCESTER, MA 01655 (Reprint); UNIV
 MASSACHUSETTS, MED CTR, DEPT MED, WORCESTER, MA 01655;
 UNIV MASSACHUSETTS, MED CTR, DEPT BIOCHEM, WORCESTER, MA
 01655; UNIV MASSACHUSETTS, MED CTR, DEPT MOL BIOL,
 WORCESTER, MA 01655; KYUSHU UNIV, MED INST BIOREGULAT,
 DEPT SURG, BEPPU, OITA, JAPAN

COUNTRY OF AUTHOR: USA; JAPAN
SOURCE: INTERNATIONAL JOURNAL OF ONCOLOGY, (MAY 1999)
Vol. 14, No. 5, pp. 927-934.
Publisher: PROFESSOR D A SPANDIDOS, 1, S MERKOURI ST,
EDITORIAL OFFICE, ATHENS 116 35, GREECE.
ISSN: 1019-6439.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Differential cDNA displays between hepatocellular carcinoma and adjacent non-malignant tissues have previously detected a PCR product, hIRH (human intercrine reduced in hepatomas), equivalent to SDF1 alpha/PBSF whose mRNA was lost from human hepatocellular carcinoma and other malignant and pre-malignant samples and malignant cell lines. There are no reports to date of the mRNA status of the receptor for hIRH/SDF1 alpha/PBSF, CXCR4 in malignant tissues. We report here that there is a reduction in the mRNA expression of CXCR4 in hepatocellular carcinoma as estimated by Northern blot and RT-PCR and compared to the adjacent non-malignant tissue. The average (mean +/- SD) tumor/normal ratio for CXCR4 mRNA expression, determined by RT-PCR, was 0.65+/-0.36 in 10 pairs of hepatocellular carcinomas. There was no consistent loss of CXCR4 mRNA expression in a range of malignant cell lines. The 3'-non-coding region

of hIRH, had typical early response gene element sequences. Despite the presence of these 3'-elements there was no induction of hIRH gene expression in human lung carcinoma A549 cells by tumor necrosis factor alpha, interleukin-2, lipopolysaccharide or phorbol myristic acetate, nor in human melanoma cell line SB-2 by UV irradiation, under conditions which induced the homologue CXCR4 intercrine IL-8 expression. Furthermore, there was no induction of hIRH gene expression, but rather a suppression, upon serum or cytokine addition to serum-deprived fibroblast cell lines, to an in vitro mouse bone marrow preparation, and to monocytic cell line THP-1.

L21 ANSWER 3 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1999:310927 SCISEARCH
THE GENUINE ARTICLE: 187GQ
TITLE: CXCR4 mRNA expression in colon, esophageal and gastric cancers and hepatitis C infected liver
AUTHOR: Mitra P; Shibuta K; Mathai J; Shimoda K; Banner B F; Mori M; Barnard G F (Reprint)
CORPORATE SOURCE: UNIV MASSACHUSETTS, MED CTR, DIV DIGEST DIS & NUTR S6 737,

55 LAKE AVE N, WORCESTER, MA 01655 (Reprint); UNIV MASSACHUSETTS, MED CTR, DIV DIGEST DIS & NUTR S6 737, WORCESTER, MA 01655; UNIV MASSACHUSETTS, MED CTR, DEPT PATHOL, WORCESTER, MA 01655; KYUSHU UNIV, MED INST BIOREGULAT, DEPT SURG, BEPPU, OITA, JAPAN

COUNTRY OF AUTHOR: USA; JAPAN
SOURCE: INTERNATIONAL JOURNAL OF ONCOLOGY, (MAY 1999)
Vol. 14, No. 5, pp. 917-925.
Publisher: PROFESSOR D A SPANDIDOS, 1, S MERKOURI ST,
EDITORIAL OFFICE, ATHENS 116 35, GREECE.
ISSN: 1019-6439.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English

REFERENCE COUNT: 49

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We have recently demonstrated by Northern blot and RT-PCR that the mRNA

expression of the alpha-chemokine hIRH/SDF-1 alpha is reduced in hepatocellular carcinoma (HCC), several digestive tract cancers and premalignant colon adenomas, and that its receptor CXCR4 mRNA expression is reduced in HCC. Here we investigate the expression of CXCR3 mRNA expression in several digestive tract cancers and hepatitis C viral (HCV) infected liver, a premalignant condition. There was no difference in the CXCR4 mRNA expression in colon, esophageal or gastric cancers compared to non-cancerous tissues. This is significantly different from the reduced expression we have seen with hepatocellular carcinoma ($p < 0.05$). To better refine regional tumor or hepatic cytokine mRNA analysis within a biopsy sample we describe a micro-isolation technique for RNA extraction from portal and triad areas of liver biopsies of other small malignant or non-malignant biopsy samples suitable for use in RT-PCR and differential display reactions. In HCV liver biopsies, the expression of hIRH and its receptor CXCR4 mRNA, corrected for G3PDH, was not significantly different from that of control non-HCV (steatosis) biopsies. CXCR3 is expressed on leukocytes and its expression was predicted to correlate with hepatic inflammation. CXCR4 receptor mRNA expression did correlate significantly with that of its ligand hIRH/SDF-1 alpha ($p = 0.001$), and with the severity of fibrosis ($p < 0.05$), but not with portal inflammation ($p < 0.10$), piecemeal necrosis ($p < 0.10$), lobular inflammation ($p > 0.10$), the presence of lymphoid aggregates ($p > 0.10$), or the total histological activity index ($p = 0.07$). There was no difference in expression of hIRH or CXCR3 between responders and non-responders to interferon (IFN) treatment, while as a control, the responder group of patients did show a higher expression of IFN alpha receptor than the non-responder group ($p = 0.05$).

L21 ANSWER 4 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 1999:62142 SCISEARCH

THE GENUINE ARTICLE: 155BH

TITLE: mRNA differential display of
2-amino-1-methyl-6-phenylinlidazo[4,5-b]pyridine-induced
rat mammary gland tumors

AUTHOR: RobertsThomson S J; Snyderwine E G (Reprint)

CORPORATE SOURCE: NCI, CHEM CARCINOGENESIS SECT, EXPT CARCINOGENESIS LAB,
DIV BASIC SCI, BLDG 37, ROOM 3C28, BETHESDA, MD 20892
(Reprint); NCI, CHEM CARCINOGENESIS SECT, EXPT
CARCINOGENESIS LAB, DIV BASIC SCI, BETHESDA, MD 20892;
UNIV QUEENSLAND, SCH PHARM, BRISBANE, QLD, AUSTRALIA

COUNTRY OF AUTHOR: USA; AUSTRALIA

SOURCE: BREAST CANCER RESEARCH AND TREATMENT, (SEP 1998)
Vol. 51, No. 2, pp. 99-107.
Publisher: KLUWER ACADEMIC PUBL, SPUIBOULEVARD 50, PO BOX
17, 3300 AA DORDRECHT, NETHERLANDS.
ISSN: 0167-6806.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE; CLIN

LANGUAGE: English

REFERENCE COUNT: 49

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The mRNA differential display technique was used to compare mRNAs between normal mammary gland and turner-derived epithelial cells from

female Sprague-Dawley rat mammary gland tumors induced by the heterocyclic amine 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) and promoted by a high-fat diet (23.5% corn oil). Two genes, beta-casein and transferrin, were identified as differentially expressed. The expression of these genes was examined across a bank of rat mammary gland tumors derived from animals on a low-fat diet (5% corn oil) or the high-fat diet.

Carcinomas had over a 10- and 50-fold lower expression of beta-casein and transferrin, respectively than normal mammary gland. In addition, carcinomas from animals on the high-fat diet showed on average a 5-fold higher expression of beta-casein, and transferrin than carcinomas from animals on the low-fat diet. The results indicate the process of mammary gland tumorigenesis alters the expression of certain genes in the mammary gland, and that the level of dietary fat further modulates the expression of these genes.

L21 ANSWER 5 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:660816 SCISEARCH
THE GENUINE ARTICLE: 113GY
TITLE: Messenger **RNA** differential display
reverse-transcriptase-polymerase-chain-reaction analysis
of a progestogen-suppressive gene in a human endometrial-
cancer cell line
AUTHOR: Sakata M (Reprint); Kurachi H; Morishige K; Ogura K;
Yamaguchi M; Nishio Y; Ikegami H; Miyake A; Murata Y
CORPORATE SOURCE: OSAKA UNIV, SCH MED, DEPT OBSTET & GYNECOL, 2-2
YAMADAOKA,
SUITA, OSAKA 565, JAPAN (Reprint)
COUNTRY OF AUTHOR: JAPAN
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (25 SEP 1998)
Vol. 78, No. 1, pp. 125-129.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605
THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0020-7136.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 25

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Progestogen suppresses the progression of endometrial cancer and has
an important effect on the secretory change of human endometrium. We
characterized the progestogen-induced alterations of gene expression in a
human endometrial-cancer cell line using a mRNA differential-display
reverse-transcriptase-polymerase-chain-reaction (DDRT-PCR) method. After
5-day incubation of Ishikawa endometrial-cancer cells, with or without
100 nM meduoxypregesterone acetate (MPA), total RNA was isolated from
confluent cells. We identified 8 candidate genes by mRNA differential
display by screening up to approximately 3,000 mRNA species. Among these,
2 genes named T21A and T21B showed a decrease in mRNA by MPA treatment
when analyzed by Northern blot. Nucleotide sequence showed that clone
T21A was part of human mitochondrial shore-chain enoyl-CoA hydratase cDNA. The
other clone, T21B, showed no homology with any known nucleotide
sequences.
Northern-blot analysis using T21A and T21B clones as probes showed a

decrease in mRNA in human endometrium from the luteal stage, with high serum estradiol and progesterone levels, as compared with that from the early follicular stage, with low serum estradiol and progesterone levels, and that from the pre-ovulatory stage with high serum estradiol and low progesterone levels. These findings suggest that mRNA DDRT-PCR could be used to identify the candidate genes regulated by progestogen in human endometrial cancer and in normal human endometrium. (C) 1998 Wiley-Liss, Inc.

L21 ANSWER 6 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 1998:340997 SCISEARCH
 THE GENUINE ARTICLE: ZK485
 TITLE: Identification of a differential expression of two **cDNAs** between malignant **mesothelioma** and normal mesothelial cells using the **RNA** fingerprint method
 AUTHOR: Frank S (Reprint); vonSpecht B U; Sendt W; Farthmann E H; Hirsch T
 CORPORATE SOURCE: UNIV FREIBURG, DEPT GEN SURG, HUGSTETTER STR 55, D-79106 FREIBURG, GERMANY (Reprint)
 COUNTRY OF AUTHOR: GERMANY
 SOURCE: TUMOR BIOLOGY, (MAY-JUN 1998) Vol. 19, No. 3, pp. 153-159.
 Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
 ISSN: 1010-4283.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: English
 REFERENCE COUNT: 22

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The RNA fingerprint method was used to identify mRNAs that were differentially expressed during the development of human mesothelial cell cancer. We report the isolation of two differentially expressed clones. One clone was expressed in the metastatic mesothelioma cell line M1A and in the malignant mesothelioma cell line M1 and downregulated in normal mesothelial cells. M1 and M1A were derived from a primary and metastatic tumor of the same patient. The other clone was only expressed in normal mesothelial cells. The different expression pattern was confirmed by Northern blot analysis. One clone had a striking sequence homology to the Soares pregnant uterus NbHPU homo sapiens cDNA clone. The other clone contained a high sequence homology to the human mRNA for ORF. The biological function of the corresponding genes is unknown. The specificity of expression of the two sequence tags was further examined on different cancer cell lines and normal tissues.

L21 ANSWER 7 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 1998:235081 SCISEARCH
 THE GENUINE ARTICLE: ZB892
 TITLE: Reduced lysyl oxidase messenger **RNA** levels in experimental and human prostate **cancer**
 AUTHOR: Ren C Z; Yang G; Timme T L; Wheeler T M; Thompson T C (Reprint)
 CORPORATE SOURCE: BAYLOR COLL MED, SCOTT DEPT UROL, 6560 FANNIN, SUITE 2100, HOUSTON, TX 77030 (Reprint); BAYLOR COLL MED, SCOTT DEPT UROL, HOUSTON, TX 77030; BAYLOR COLL MED, DEPT PATHOL,

HOUSTON, TX 77030; BAYLOR COLL MED, DEPT CELL BIOL,
HOUSTON, TX 77030; BAYLOR COLL MED, DEPT RADIOLOG, HOUSTON,
TX 77030

COUNTRY OF AUTHOR: USA
SOURCE: CANCER RESEARCH, (15 MAR 1998) Vol. 58, No. 6,
pp. 1285-1290.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806,
BIRMINGHAM, AL 35202.
ISSN: 0008-5472.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 27

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To identify genes associated with prostate cancer progression, we developed a strategy involving the use of differential display PCR and a panel of genetically matched primary tumor-and metastasis-derived mouse prostate cancer cell lines. We analyzed sequences that were differentially stimulated by transforming growth factor-beta 1 in primary tumor-versus metastasis-derived cell lines, based on our previous studies indicating that acquisition of differential responses to this growth factor could result in phenotypic traits that facilitate tumor metastasis from specific cell clones within the primary tumor. Using this system, we isolated and sequenced a cDNA fragment that encoded mouse lysyl oxidase (LO) and was induced by transforming growth factor-beta 1d in primary tumor but not in metastasis-derived cells. Northern blotting analysis revealed increased LO expression in a panel of primary tumor cell lines but significantly reduced or nondetectable expression in their matched metastatic counterparts. Further in situ hybridization analysis revealed LO expression in normal mouse prostate epithelium but, in most cases, progressive loss of expression in primary prostate cancer and associated metastatic lesions. Importantly, in situ hybridization studies of normal human prostate and prostate malignancies revealed a similar loss of expression during progression to metastasis. The progressive loss of LO expression during prostate cancer progression provides information that may increase our understanding of the mechanisms that underlie this disease. In addition, LO may provide a useful molecular marker and/or establish a novel therapeutic target for prostate cancer.

L21 ANSWER 8 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 97:435194 SCISEARCH
THE GENUINE ARTICLE: XC009
TITLE: Expression of a novel mRNA in human head and neck squamous cell carcinoma cells
AUTHOR: Werner J A (Reprint); Gorogh T; Lippert B M; Gottschlich S; Heidorn K; Folz B J; Rudert H
CORPORATE SOURCE: CHRISTIAN ALBRECHTS UNIV KIEL, DEPT OTORHINOLARYNGOL HEAD & NECK SURG, ARNOLD HELLER STR 14, D-24105 KIEL, GERMANY (Reprint); CHRISTIAN ALBRECHTS UNIV KIEL, DEPT HEMATOPATHOL, D-24098 KIEL, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: JOURNAL OF CLINICAL PATHOLOGY-MOLECULAR PATHOLOGY, (APR 1997) Vol. 50, No. 2, pp. 82-86.
Publisher: BRITISH MED JOURNAL PUBL GROUP, BRITISH MED ASSOC HOUSE, TAVISTOCK SQUARE, LONDON, ENGLAND WC1H 9JR.

ISSN: 1355-2910.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 33

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Aims-The differential display reverse transcription polymerase chain reaction (DDRT-PCR) technique was used to search for differences between the mRNA expression profiles of squamous cell carcinoma (SCC) cell lines established from head and neck tumours and normal keratinocytes from the mucosa of the upper aerodigestive tract.

Methods-Total RNA prepared from both cell types was reverse transcribed into cDNA then amplified in a PCR mixture. To compare the electrophoretic patterns, mRNAs were amplified by nested PCR using specific oligonucleotides. Additionally, using labelled cDNA probes, northern hybridisation was carried out on three cancer cell lines of different origin, a biopsy from a parotid gland pleomorphic adenoma, healthy mucosa, and keratinocytes.

Results-Comparison of the separated bands revealed a fragment with a differential expression pattern in the SCC cells. This cloned sequence of a 336 base pair mRNA fragment exhibited no significant homology with known

transcripts. Additionally, after amplification and sequencing of the 3' end of the fragment no homology with a known human gene sequence was found. However, low homology with a genomic sequence of a nematode was found. Northern hybridisation confirmed the selective expression of this fragment in SCC cells versus the cancer cell lines of different origin, the biopsy of the pleomorphic adenoma, keratinocytes, and healthy mucosa.

Conclusions-This is the first differentially expressed human genome transcript of squamous cell carcinoma of the head and neck identified by DDRT-PCR. It may prove useful, in the future, to characterise this tumour type.

L21 ANSWER 9 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 97:164686 SCISEARCH

THE GENUINE ARTICLE: WH815

TITLE: Identification of a breast **cancer**-specific gene, BCSG1, by direct differential **cDNA** sequencing

AUTHOR: Ji H J; Liu Y L E; Jia T L; Wang M S; Liu J W; Xiao G W; Joseph B K; Rosen C; Shi Y E (Reprint)

CORPORATE SOURCE: ALBERT EINSTEIN COLL MED, LONG ISL JEWISH MED CTR, SCHNEIDER CHILDRENS HOSP, PEDIAT RES CTR, NEW HYDE PK, NY 11042 (Reprint); ALBERT EINSTEIN COLL MED, LONG ISL

JEWISH

MED CTR, SCHNEIDER CHILDRENS HOSP, PEDIAT RES CTR, NEW HYDE PK, NY 11042; ALBERT EINSTEIN COLL MED, LONG ISL JEWISH MED CTR, SCHNEIDER CHILDRENS HOSP, PEDIAT RES CTR, NEW HYDE PK, NY 11042; HUMAN GENOME SCI INC, ROCKVILLE,

MD

20850; MT STATES MED RES INST, BOISE, ID 83702; DEPT VET AFFAIRS MED CTR, BOISE, ID 83702

COUNTRY OF AUTHOR: USA

SOURCE: CANCER RESEARCH, (15 FEB 1997) Vol. 57, No. 4, pp. 759-764.

Publisher: AMER ASSOC CANCER RESEARCH, PUBLIC LEDGER

BLDG,

SUITE 816, 150 S. INDEPENDENCE MALL W., PHILADELPHIA, PA
19106.

ISSN: 0008-5472.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 49

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A high-throughput direct-differential cDNA sequencing approach was employed to identify genes differentially expressed in normal breast as compared with breast cancer. Approximately 6000 expressed sequence tags (ESTs) from cDNA libraries of normal breast and breast carcinoma were selected randomly and subjected to EST-sequencing analysis. The relative expression levels of more than 2000 unique EST groups were quantitatively compared in normal versus cancerous breast. Of many putative differentially expressed genes, a breast cancer-specific gene, BCSG1, which was expressed in high abundance in a breast cancer cDNA library but scarcely in a normal breast cDNA library, was identified as a putative breast cancer marker. In situ hybridization analysis demonstrated stage-specific BCSG1 expression as follows: BCSG1 was undetectable in normal or benign breast lesions, showed partial expression in ductal carcinoma in situ, but was expressed at an extremely high level in advanced infiltrating breast cancer. The predicted amino acid sequence of BCSG1 gene has a significant sequence homology to the non-amyloid beta protein fragment of the Alzheimer's disease amyloid protein. BCSG1 overexpression may indicate breast cancer malignant progression from benign breast or in situ carcinoma to the highly infiltrating carcinoma.

L21 ANSWER 10 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 96:866614 SCISEARCH

THE GENUINE ARTICLE: VT536

TITLE: Increased expression of T-plastin gene in
cisplatin-resistant human **cancer** cells:
Identification by mRNA differential display

AUTHOR: Hisano T; Ono M; Nakayama M; Naito S; Kuwano M; Wada M
(Reprint)

CORPORATE SOURCE: KYUSHU UNIV, SCH MED, DEPT BIOCHEM, FUKUOKA 81282, JAPAN
(Reprint); KYUSHU UNIV, SCH MED, DEPT BIOCHEM, FUKUOKA
81282, JAPAN; KYUSHU UNIV, SCH MED, DEPT UROL, FUKUOKA
81282, JAPAN

COUNTRY OF AUTHOR: JAPAN

SOURCE: FEBS LETTERS, (11 NOV 1996) Vol. 397, No. 1, pp.
101-107.

Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.

ISSN: 0014-5793.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The cellular resistance to the potent anticancer agent
cis-diamminedichloroplatinum(II) (cisplatin) is thought to be mediated by
multiple mechanisms. The technique of differential display of mRNAs was
applied to various cisplatin-resistant cell lines and the corresponding
parental sensitive human bladder, prostatic, and head and neck cancer
cells in order to identify genes that underlie cisplatin resistance.
Twenty-four clones were confirmed by Northern blot analysis to be

expressed differentially between resistant and the corresponding sensitive cells. Partial DNA sequences of the eight clones that showed a threefold or greater increase in expression in either the resistant cells (seven clones) or sensitive cells (one clone) revealed that two were derived from the T-plastin gene and one from the tissue factor gene. The abundance of T-plastin mRNA in cisplatin-resistant T24/DDP10 cell was similar to 12 times that in the parental T24 cells. Transfection of T24/DDP10 cells with a vector encoding full-length T-plastin antisense RNA demonstrated that reduced T-plastin expression was associated with increased sensitivity to cisplatin. These results are consistent with the hypothesis that several mechanisms participate cooperatively in the acquisition of cisplatin resistance in human cancer.

L21 ANSWER 11 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 96:553812 SCISEARCH
 THE GENUINE ARTICLE: UY063
 TITLE: IDENTIFICATION BY DIFFERENTIAL DISPLAY OF A MESSENGER-RNA SPECIFICALLY INDUCED BY 12-O-TETRADECANOYLPHORBOL-13-ACETATE (TPA) IN T84 HUMAN COLON-CARCINOMA CELLS
 AUTHOR: CAFFERATA E G; GONZALEZGUERRICO A M; PIVETTA O H; SANTACOLOMA T A (Reprint)
 CORPORATE SOURCE: FDN CAMPOMAR, INST INVEST BIOQUIM, AV PATRICIAS ARGENTINAS
 435, RA-1405 BUENOS AIRES, DF, ARGENTINA (Reprint); FDN CAMPOMAR, INST INVEST BIOQUIM, RA-1405 BUENOS AIRES, DF, ARGENTINA; INST NAEL GENET MED, BUENOS AIRES, DF, ARGENTINA
 COUNTRY OF AUTHOR: ARGENTINA
 SOURCE: CELLULAR AND MOLECULAR BIOLOGY, (JUL 1996) Vol. 42, No. 5, pp. 797-804.
 ISSN: 0145-5680.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 16

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB 12-o-Tetradecanoylphorbol-13-Acetate (TPA) down-regulates the expression of the gene responsible for cystic fibrosis (CFTR). To understand the mechanism by which TPA down-regulates CFTR, we decided to study genes specifically induced by this phorbol ester in T84 human colon carcinoma cells, which highly express CFTR, using differential display. Several strategies that allowed us to overcome false-positive reactions

in differential displays are described. We have detected different cDNAs obtained from mRNAs specifically induced by TPA. A cDNA fragment corresponding to a mRNA of approximately 2.2 kb was sequenced. Part of this sequence has been reported by others in GenBank and corresponds to a cDNA from a human lung library. The function is unknown and does not have any significant homology with other sequences. It is expressed after two hrs in T84 cells treated with TPA, reaching a maximum response by four hrs. The dose-response curve shows increased mRNA levels starting at 10 ng/ml of TPA and reaching a maximum by 50 ng/ml TPA (10-fold stimulation over control). This mRNA shows a rapid and large response to TPA and it might be involved in the differentiation of T84 cells induced by TPA.

L21 ANSWER 12 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 96:552791 SCISEARCH

THE GENUINE ARTICLE: UY023

TITLE: IDENTIFICATION OF MESSENGER-RNAS DIFFERENTIALLY
EXPRESSED IN QUIESCENCE OR IN LATE G1 PHASE OF THE
CELL-CYCLE IN HUMAN BREAST-CANCER CELLS BY USING
THE DIFFERENTIAL DISPLAY METHOD

AUTHOR: ALPAN R S; SPARVERO S; PARDEE A B (Reprint)

CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST D810, DIV
CELL GROWTH & REGULAT, 44 BINNEY ST, BOSTON, MA, 02115
(Reprint); HARVARD UNIV, SCH MED, DANA FARBER CANC INST
D810, DIV CELL GROWTH & REGULAT, BOSTON, MA, 02115;
HARVARD UNIV, SCH MED, DEPT BIOL CHEM & MOLEC PHARMACOL,
BOSTON, MA, 02115

COUNTRY OF AUTHOR: USA

SOURCE: MOLECULAR MEDICINE, (JUL 1996) Vol. 2, No. 4,
pp. 469-478.

ISSN: 1076-1551.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE; CLIN

LANGUAGE: ENGLISH

REFERENCE COUNT: 23

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background: The decision for a cell to enter the DNA synthesis (S)
phase of the cell cycle or to arrest in quiescence is likely to be
determined by genes expressed in the late G1 phase, at the restriction
point. Loss of restriction point control is associated with malignant
cellular transformation and cancer. For this reason, identifying genes
that are differentially expressed in late G1 phase versus quiescence is
important for understanding the molecular basis of normal and malignant
growth.

Materials and Methods: The differential display (DD) method detects
mRNA species that are different between sets of mammalian cells, allowing
their recovery and cloning of the corresponding cDNAs. Using this
technique, we compared mRNAs from synchronized human breast cancer cells
(21PT) in quiescence and in late G1.

Results: Six mRNAs differentially expressed in late G1 or in
quiescence

were identified. One mRNA expressed 10 hr after serum induction showed
99%

homology to a peptide transporter involved in antigen presentation of the
class I major histocompatibility complex (TAP-1) mRNA. Another mRNA
expressed specifically in quiescence and down-regulated 2 hr following
serum induction showed 98% homology to human NADP(+)-dependent
cytoplasmic

malic enzyme (EC1.1.1.40) mRNA, which is an important enzyme in fatty acid
synthesis and lipogenesis. Three others showed high homology to different
mRNAs in the GeneBank, corresponding to genes having unknown functions.
Finally, one mRNA revealed no significant homology to known genes in the
GeneBank.

Conclusions: We conclude that DD is an efficient and powerful method
for the identification of growth-related genes which may have a role in
cancer development.

L21 ANSWER 13 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 95:846741 SCISEARCH

THE GENUINE ARTICLE: TH623

TITLE: IDENTIFICATION OF A TRANSPOSON-RELATED **RNA**
DOWN-REGULATED BY RETINOIC ACID IN EMBRYONAL
CARCINOMA AND EMBRYONIC STEM-CELLS
AUTHOR: HIROSE T; AUSTIN S J; JETTEN A M (Reprint)
CORPORATE SOURCE: NIEHS, CELL BIOL SECT, PULM PATHOBIOL LAB, RES TRIANGLE
PK, NC, 27709 (Reprint); NIEHS, CELL BIOL SECT, PULM
PATHOBIOL LAB, RES TRIANGLE PK, NC, 27709
COUNTRY OF AUTHOR: USA
SOURCE: EXPERIMENTAL CELL RESEARCH, (DEC 1995) Vol. 221,
No. 2, pp. 294-300.
ISSN: 0014-4827.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The differential display polymerase chain reaction was employed to
identify changes in mRNA expression during retinoic acid-induced
differentiation in embryonal carcinoma PCC4.azalR cells, In this study,

we report on one cDNA, EC1, that was identified by this method, EC-1 encodes
a 0.6-kb mRNA that is present in PCC4.azalR cells and down-regulated by
retinoic acid, Sequence analysis revealed that EC-1 exhibits a 47%
identity with the early transposon RNA ETn and does not contain a long
open reading frame, EC-1 mRNA expression was reduced by 50% after 24 h of
treatment with 10 nM retinoic acid and was undetectable after 48 h,
Down-regulation of EC-1 mRNA was observed at retinoic acid concentrations
as low as 0.1 nM. EC-1 was found to be expressed in several other
embryonal carcinoma cell lines as well as in embryonic stem cells but was
undetectable in differentiated cell types obtained after RA treatment,
Northern blot analysis using RNA from multiple mouse tissues demonstrated
that the expression of EC-1 is restricted to the testis, Treatment of
PCC4.azalR cells with an RAR-selective agonist also repressed the
expression of EC-1 mRNA while treatment with an RXR-selective agonist
reduced EC-1 expression slightly. The RAR alpha-specific antagonist Ro
41-5253 had little effect on the downregulation of EC-1 by retinoic acid,
Our observations indicate that the repression of EC-1 is associated with
the induction of differentiation in embryonal carcinoma and embryonic

stem
cells and involves an RAR-activated signaling pathway. (C) 1995 Academic
Press, Inc.

L21 ANSWER 14 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 95:745156 SCISEARCH

THE GENUINE ARTICLE: RU338

TITLE: IDENTIFICATION OF DIFFERENTIALLY EXPRESSED MESSENGER-
RNAS DURING NEURONAL DIFFERENTIATION OF P19
EMBRYONAL **CARCINOMA**-CELLS

AUTHOR: SUZUKI Y (Reprint); WANAKA A; TOHYAMA M; TAKAGI T
CORPORATE SOURCE: OSAKA UNIV, SCH MED, DEPT MOLEC NEUROBIOL TANABE, 2-2
YAMADAOKA, SUITA, OSAKA 565, JAPAN (Reprint); OSAKA UNIV,
SCH MED, DEPT ANAT & NEUROSCI, SUITA, OSAKA 565, JAPAN

COUNTRY OF AUTHOR: JAPAN
SOURCE: NEUROSCIENCE RESEARCH, (AUG 1995) Vol. 23, No.
1, pp. 65-71.
ISSN: 0168-0102.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE

LANGUAGE: ENGLISH
REFERENCE COUNT: 22

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To understand the basic mechanisms underlying neuronal differentiation,
we have attempted to isolate differentially expressed genes, which may play a key role in this complex process, from neuronal differentiating
P19 embryonal carcinoma cells. RNA fingerprinting by the arbitrarily primed PCR (RAP) method was adapted to detect such differentially expressed genes
during P19 neuronal differentiation. Using this method with some modifications, we successfully cloned seven cDNA fragments which were expressed differentially within the first 48 h after 1 μ M retinoic acid (RA) treatment, which ultimately induces neuronal differentiation. Comparison of the partial nucleotide sequences of these clones with sequences in DNA databases indicated that one of these clones was identical to a region of the mouse Oct-3 gene, which has been shown to be dramatically repressed by RA. Two clones were highly homologous to the human profilinII and leucine-rich protein genes. The other four clones were not closely related to any sequences in the databases. Except for the
Oct-3 gene, the other six genes isolated here have not been reported previously as RA-regulated genes. RAP is, thus, a promising method for identification of novel and potentially important genes which are differentially regulated during neuronal differentiation.

L21 ANSWER 15 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 95:486111 SCISEARCH

THE GENUINE ARTICLE: RH966

TITLE: RNA GENETICS OF BREAST-CANCER - MASPIN
AS PARADIGM

AUTHOR: SAGER R (Reprint); SHENG S; ANISOWICZ A; SOTIROPOULOU G;
ZOU Z; STENMAN G; SWISSHELM K; CHEN Z; HENDRIX M J C;
PEMBERTON P; RAFIDI K; RYAN K

CORPORATE SOURCE: DANA FARBER CANC INST, BOSTON, MA, 02115 (Reprint);
GOTHENBURG UNIV, SAHLGRENSKA HOSP, DEPT PATHOL, CANC

GENET

LAB, S-41345 GOTHENBURG, SWEDEN; UNIV WASHINGTON, DEPT
PATHOL, SEATTLE, WA, 98195; ST LOUIS UNIV, CARDINAL
GLENNON CHILDRENS HOSP, SCH MED, PEDIAT RES INST, ST
LOUIS, MO, 63110; LXR BIOTECHNOL INC, RICHMOND, CA, 94804

COUNTRY OF AUTHOR: USA; SWEDEN

SOURCE: COLD SPRING HARBOR SYMPOSIA ON QUANTITATIVE BIOLOGY, (1994) Vol. 59, pp. 537-546.
ISSN: 0091-7451.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 40

L21 ANSWER 16 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 95:301868 SCISEARCH

THE GENUINE ARTICLE: QU879

TITLE: ISOLATION OF CDNAS THAT ARE DIFFERENTIALLY
EXPRESSED BETWEEN ANDROGEN-DEPENDENT AND
ANDROGEN-INDEPENDENT PROSTATE CARCINOMA-CELLS
USING DIFFERENTIAL DISPLAY PCR

AUTHOR: BLOK L J; KUMAR M V; TINDALL D J (Reprint)
 CORPORATE SOURCE: MAYO CLIN & MAYO FDN, DEPT UROL RES, 17 GUGGENHEIM,
 ROCHESTER, MN, 55905 (Reprint); MAYO CLIN & MAYO FDN,
 DEPT
 UROL RES, ROCHESTER, MN, 55905; MAYO CLIN & MAYO FDN,
 DEPT
 BIOCHEM MOLEC BIOL, ROCHESTER, MN, 55905
 COUNTRY OF AUTHOR: USA
 SOURCE: PROSTATE, (APR 1995) Vol. 26, No. 4, pp. 213-224
 .
 ISSN: 0270-4137.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 43

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In the development of prostate cancer there is an important transition from androgen-dependent growth (which can be treated) to androgen-independent growth (which is beyond medical control). This transition is probably accompanied by genetic changes, resulting in the activation of oncogenes or the inactivation of tumor suppressor genes. In the present manuscript, the isolation of genes that may be involved in advanced, androgen-independent prostate cancer growth is described.

Using differential display PCR, 13 cDNAs were isolated representing genes that are differentially expressed between the androgen-dependent prostate carcinoma cell line LNCaP and the androgen-independent prostate carcinoma cell lines PC-3 and DU 145. These clones were divided into four groups: androgen-responsive genes (TL5, TL25, TL32, and TL35); genes with a marked decreased expression in one of the prostate cancer cell lines (TL27); genes with a marked, increased expression in one or more of the prostate cancer cell lines (TL4, TL16, TL21, and TL22); and genes with minor (but repeatable) changes in expression between prostate cancer cell lines (TL7, TL15, TL18, and TL33). The 13 genes were analyzed for their sequence information, tissue specificity, and androgen responsiveness in order to identify genes of interest.

In summary, differential display PCR appears to provide an attractive alternative to existing molecular techniques to screen for differentially expressed genes in prostate cancer cells. (C) 1995 Wiley-Liss, Inc.

L21 ANSWER 17 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 95:209191 SCISEARCH

THE GENUINE ARTICLE: QM109

TITLE: IDENTIFICATION AND PARTIAL SEQUENCE OF A CDNA
 THAT IS DIFFERENTIALLY EXPRESSED IN HUMAN BRAIN-
TUMORS

AUTHOR: SHINOURA N; SHAMRAJ O I; HUGENHOLZ H; ZHU J G; MCBBLACK P;
 WARNICK R; TEW J J; WANI M A; MENON A G (Reprint)

CORPORATE SOURCE: UNIV CINCINNATI, COLL MED, DEPT MOLEC GENET BIOCHEM &
 MICROBIOL, 231 BETHESDA AVE, CINCINNATI, OH, 45267
 (Reprint); UNIV CINCINNATI, COLL MED, DEPT MOLEC GENET
 BIOCHEM & MICROBIOL, CINCINNATI, OH, 45267; OTTAWA GEN
 HOSP, DEPT NEUROSURG, OTTAWA, ON K1H 8L6, CANADA; HARVARD
 UNIV, BRIGHAM & WOMENS HOSP, SCH MED, DEPT NEUROSURG,
 BOSTON, MA, 02114; UNIV CINCINNATI, COLL MED, DEPT
 NEUROSURG, CINCINNATI, OH, 45267

COUNTRY OF AUTHOR: USA; CANADA

SOURCE: CANCER LETTERS, (02 MAR 1995) Vol. 89, No. 2,
 pp. 215-221.

ISSN: 0304-3835.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 14

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Differential display technique was used to identify mRNAs that are differentially expressed in malignant versus benign brain tumors. Using this method, a novel 1.4-kb long cDNA (MM1) clone was isolated and sequenced. The nucleotide and the translated amino acid sequence of MM1 cDNA clone did not show significant homology to any known sequence in the Genbank. The expression of MM1 appears to be almost eightfold higher in glioblastomas compared to low grade astrocytomas and slightly higher in malignant meningiomas than in benign meningiomas. The size of mRNA from northern analysis appears to be 7 kb, which is much higher than the size of the isolated MM1 cDNA clone. Expression of MM1 was also seen in various cell lines derived from human tumors including glioblastomas. Whereas low level expression was seen in kidney, esophagus, liver, lymph node, ovary and testis, none of the other tissues from a total of 18 different human organs showed any MM1 expression.

L21 ANSWER 18 OF 18 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 93:496221 SCISEARCH

THE GENUINE ARTICLE: LR606

TITLE: IDENTIFICATION OF DIFFERENTIALLY EXPRESSED RNA
IN HUMAN OVARIAN-CARCINOMA CELLS BY ARBITRARILY
PRIMED PCR FINGERPRINTING OF TOTAL RNAS

AUTHOR: WONG K K; MOK C H (Reprint); WELSH J T; MCCLELLAND M;
TSAO

S W; BERKOWITZ R S
CORPORATE SOURCE: BRIGHAM & WOMENS HOSP, DEPT OBSTET GYNECOL & REPROD BIOL,
GYNECOL ONCOL LAB, BOSTON, MA, 02115; CALIF INST BIOL
RES,

LA JOLLA, CA, 92037

COUNTRY OF AUTHOR: USA

SOURCE: INTERNATIONAL JOURNAL OF ONCOLOGY, (JUL 1993)
Vol. 3, No. 1, pp. 13-17.

ISSN: 1019-6439.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 22

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Using arbitrarily primed PCR fingerprinting of RNA (RAP), we have analyzed RNAs prepared from two normal ovarian surface epithelial cell cultures, two normal mesothelial cell cultures, and eight independent ovarian carcinoma cell lines. Each arbitrarily chosen primer gave a

unique

fingerprint of about 30 cDNA products. Most of the cDNA products produced by any particular primer were shared between all cell lines. However, one primer detected a cDNA PCR product that was absent in all eight ovarian carcinoma cell lines but present in all normal cell cultures. We have cloned and sequenced the DNA fragment that distinguishes normal from ovarian carcinoma cell lines. The DNA sequence has one continuous open reading frame throughout which indicates that it may be from a translated gene. Furthermore, we confirmed the differential expression of the gene

by

Northern blot analysis. We also observed that the intensity of the band
in the RNA fingerprint correlates with the expression level of the
corresponding RNA. These results further demonstrate the ability of RAP
to detect differentially expressed genes in a quantitative manner and
demonstrated the application of RAP for the detection of differential
gene expression during carcinogenesis.

=>

L1 ANSWER 1 OF 1 MEDLINE
 ACCESSION NUMBER: 93092151 MEDLINE
 DOCUMENT NUMBER: 93092151 PubMed ID: 1458489
 TITLE: Differential display and cloning of messenger RNAs from human breast cancer versus mammary epithelial cells.
 AUTHOR: Liang P; Averboukh L; Keyomarsi K; Sager R; Pardee A B
 CORPORATE SOURCE: Division of Cell Growth and Regulation, Dana-Farber Cancer Institute, Boston, Massachusetts 02115.
 CONTRACT NUMBER: GM 24571 (NIGMS)
 SOURCE: CANCER RESEARCH, (1992 Dec 15) 52 (24) 6966-8.
 Journal code: CNF; 2984705R. ISSN: 0008-5472.
 PUB. COUNTRY: United States
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 OTHER SOURCE: GENBANK-S50179; GENBANK-S50182; GENBANK-S50184; GENBANK-S50185; GENBANK-S72766; GENBANK-S72767; GENBANK-S72768; GENBANK-S72769; GENBANK-S72771; GENBANK-X63098
 ENTRY MONTH: 199301
 ENTRY DATE: Entered STN: 19930129
 Last Updated on STN: 19930129
 Entered Medline: 19930108
 AB Identification of the genes that are specifically expressed in tumor cells but not in normal cells (oncogenes), or vice versa (tumor suppressor genes), is important for understanding the molecular basis of cancer. The differential display technique was applied to compare mRNAs from normal and tumor-derived human mammary epithelial cells, cultured under the same conditions. Complementary DNA fragments corresponding to several apparently differentially expressed mRNAs were recovered and sequenced. They exhibit characteristics of the 3' end of eukaryotic mRNA, as predicted by the method. A complementary DNA fragment seen only in the normal cell was used as a probe to isolate its corresponding complementary DNA clone from a library. Northern analysis confirmed its differential expression. Thus, this method can be used for detecting, cloning, and sequencing of genes that are unique to a host of biological and disease processes.

=> file scisearch

L20 ANSWER 3 OF 6 MEDLINE DUPLICATE 3
 ACCESSION NUMBER: 1998389283 MEDLINE
 DOCUMENT NUMBER: 98389283 PubMed ID: 9723890
 TITLE: Identification of a second **stanniocalcin** cDNA in mouse and human: **stanniocalcin 2**.
 AUTHOR: **Chang A C; Reddel R R**
 CORPORATE SOURCE: Children's Medical Research Institute, Sydney, NSW, Australia.
 SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1998 Jun 25) 141 (1-2) 95-9.
 Journal code: E69; 7500844. ISSN: 0303-7207.
 PUB. COUNTRY: Ireland
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 OTHER SOURCE: GENBANK-AF055460; GENBANK-AF056244
 ENTRY MONTH: 199811
 ENTRY DATE: Entered STN: 19990106
 Last Updated on STN: 20000303
 Entered Medline: 19981112
 AB It has recently been shown that mammals contain a gene encoding a protein that is related to **stanniocalcin** (STC), a hormone previously considered to be present only in bony fish where it plays a major role in Ca²⁺ homeostasis. Here we report the identification in the mouse and human of a cDNA, STC-2, that shows significant similarity to the first mammalian STC (which we now rename STC-1). Northern analysis revealed that mammalian STC-2, like STC-1, is expressed in a wide variety of tissues. In contrast to STC-1, the predicted amino acid sequence of STC-2 contains a cluster of histidine residues in the C-terminal portion of the protein, which suggests that STC-2 may interact with metal ions.

L20 ANSWER 4 OF 6 MEDLINE DUPLICATE 4

L7 ANSWER 4 OF 9

MEDLINE

DUPLICATE 3

ACCESSION NUMBER: 1999145369 MEDLINE

DOCUMENT NUMBER: 99145369 PubMed ID: 10022771

TITLE: Molecular cloning and characterization of
stanniocalcin-related protein.

AUTHOR: DiMattia G E; Varghese R; Wagner G F

CORPORATE SOURCE: Department of Oncology, The University of Western Ontario,
The London Regional Cancer Centre, Canada..

dimattia@julian.uwo.ca

SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1998 Nov 25)
146 (1-2) 137-40.

Journal code: E69; 7500844. ISSN: 0303-7207.

PUB. COUNTRY: Ireland

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199905

ENTRY DATE: Entered STN: 19990517

Last Updated on STN: 19990517

Entered Medline: 19990503

AB **Stanniocalcin** (STC) is a glycoprotein hormone first discovered
in fish and recently identified in humans and mice. In this report we
have

described the cloning of an STC-like cDNA, designated here as STC related
protein (STCrP). Human STCrP (hSTCrP) cDNAs were isolated as expressed
sequence tags (ESTs) and predicted a polypeptide of 302 amino acids, with
58% homology to human STC (hSTC). Ten of the eleven 1/2 cysteine
residues

that in STC allow for dimerization of monomeric subunits were conserved
in

hSTCrP. By Northern analysis, three hSTCrP mRNAs were detected and were
most abundant in pancreas, spleen and kidney as well as a variety of
different transformed cell lines. The high degree of sequence homology
suggests that STC and STCrP may have been derived from a common ancestral
gene..

L10 ANSWER 15 OF 19 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:250622 CAPLUS

DOCUMENT NUMBER: 133:133356

TITLE: Assessment of Stanniocalcin-1 mRNA as a molecular marker for micrometastases of various human **cancers**

AUTHOR(S): Fujiwara, Yoshiyuki; Sugita, Yurika; Nakamori, Shoji; Miyamoto, Atsushi; Shiozaki, Ken; Nagano, Hiroaki; Sakon, Masato; Monden, Morito

CORPORATE SOURCE: Department of Surgery and Clinical Oncology, Osaka University, Osaka, 565-0871, Japan

SOURCE: Int. J. Oncol. (2000), 16(4), 799-804

CODEN: IJONES; ISSN: 1019-6439

PUBLISHER: International Journal of Oncology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We assessed Stanniocalcin-1 (STC-1) mRNA expression in human normal tissues, various types of human **cancer** cell lines, and **cancer** tissues obtained during surgery. Using a reverse transcriptase-polymerase chain reaction (RT-PCR) assay developed to detect

STC-1 mRNA, the transcripts were detected in 20 out of 21 **cancer** cell lines and in all **tumor** tissues from various types of **cancer**. Semi-quant. analyses with multiplex RT-PCR showed that STC-1 mRNA tended to be enhanced in **cancer** tissues of hepatocellular **carcinoma** and colorectal **cancer** compared to background **cancer**-free tissues. Anal. of blood samples obtained from 11 patients with hepatocellular **carcinoma** before, after and during hepatectomy showed STC-1 mRNA expression in 8 out

of 11 patients at least one time. Normal donor blood samples (n=31) were all-neg. for STC-1 mRNA expression. Our results indicate that STC-1 mRNA might be a useful mol. marker for detection of **tumor** cells in blood from patients with various types of **malignancies**.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS

FORMAT RECORD. ALL CITATIONS AVAILABLE IN THE RE

L10 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:19480 CAPLUS

DOCUMENT NUMBER: 132:74509

TITLE: A method and a kit for diagnosing leukemia using stanniocalcin as a **tumor** marker

INVENTOR(S): Koide, Yoshio; Igarashi, Akira; Fu, Jianguo; Takano, Shouichi; Sasaki, Takeshi

PATENT ASSIGNEE(S): BML K. K., Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 10 pp.

CODEN: JKXXAF

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	-----		-----	-----	-----
	JP 2000002709	A2	20000107	JP 1998-181503	19980613
AB	A convenient method is provided for accurately diagnosing leukemia using stanniocalcin as a tumor marker. Stanniocalcin is a glycoprotein specifically recognized in blood of a patient upon developing leukemia. More specifically, stanniocalcin is recognized in the blood cell fraction from which red blood cells are excluded. The occurrence of stanniocalcin is examd. either by an immunol. method using antibodies to stanniocalcin or by a genetic amplification method detecting mRNA for stanniocalcin in the blood cell fraction. A test kit including at least antibodies to stanniocalcin protein or heat-stable DNA polymerase for diagnosing leukemia is claimed. MRNA for stanniocalcin was detected with most of leukemia patients, but not with healthy persons at all. Stanniocalcin protein was detected with leukemia cells by flow cytometry using anti-stanniocalcin monoclonal antibody.				

L10 ANSWER 17 OF 19 CAPLUS COPYRIGHT 2002 ACS

L12 ANSWER 4 OF 5 JAPIO COPYRIGHT 2002 JPO

ACCESSION NUMBER: 2000-002709 JAPIO

TITLE: LEUKEMIA DETECTING METHOD AND DETECTION KIT

INVENTOR: KOIDE YOSHIO; IGARASHI AKIRA; KO TAKEKUNI; TAKANO
SHOICHI; SASAKI TAKESHI

PATENT ASSIGNEE(S): BML:KK)

PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 2000002709A		20000107	Heisei	G01N033-68

JP

APPLICATION INFORMATION

ST19N FORMAT: JP1998-181503 19980613

ORIGINAL: JP10181503 Heisei

SOURCE: PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined
Applications, Vol. 2000

AN 2000-002709 JAPIO

AB PROBLEM TO BE SOLVED: To find out a specific factor detected in a body
fluid specimen when a person contracts leukemia, and to provide a simple
and accurate diagnostic method for the leukemia using this specific
factor

as the base.

SOLUTION: A state of **stanniocalcin** of a blood corpuscle fraction
of blood, preferably a blood corpuscle fraction from which a red blood
corpuscle fraction is removed is grasped by a detection method using an
antigen-antibody reaction between an antibody to the **stanniocalcin**
and the **stanniocalcin**, detection of mRNA of the
stanniocalcin in the blood corpuscle fraction, or the like. Using
the **stanniocalcin** as an index of the leukemia diagnosis can make
the diagnosis method simple and accurate.

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WEST☐ Generate Collection

L14: Entry 1 of 2

File: JPAB

Jan 7, 2000

PUB-NO: JP02000002709A

DOCUMENT-IDENTIFIER: JP 2000002709 A

TITLE: LEUKEMIA DETECTING METHOD AND DETECTION KIT

PUBN-DATE: January 7, 2000

INVENTOR-INFORMATION:

NAME

COUNTRY

KOIDE, YOSHIO

IGARASHI, AKIRA

KO, TAKEKUNI

TAKANO, SHOICHI

SASAKI, TAKESHI

ASSIGNEE-INFORMATION:

NAME

COUNTRY

BML:KK

APPL-NO: JP10181503

APPL-DATE: June 13, 1998

INT-CL (IPC): G01N 33/68; C12N 15/02; G01N 33/53; C12P 21/08

ABSTRACT:

PROBLEM TO BE SOLVED: To find out a specific factor detected in a body fluid specimen when a person contracts leukemia, and to provide a simple and accurate diagnostic method for the leukemia using this specific factor as the base.

SOLUTION: A state of stanniocalcin of a blood corpuscle fraction of blood, preferably a blood corpuscle fraction from which a red blood corpuscle fraction is removed is grasped by a detection method using an antigen-antibody reaction between an antibody to the stanniocalcin and the stanniocalcin, detection of mRNA of the stanniocalcin in the blood corpuscle fraction, or the like. Using the stanniocalcin as an index of the leukemia diagnosis can make the diagnosis method simple and accurate.

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WEST**End of Result Set**☐ Generate Collection

L14: Entry 2 of 2

File: DWPI

Jan 7, 2000

DERWENT-ACC-NO: 2000-131570

DERWENT-WEEK: 200016

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TITLE: Diagnosis of leukemia - involves evaluating stanniocalcin present in leucocytes using antibody specific for stanniocalcin

PATENT-ASSIGNEE: BML KK (BMLBN)

PRIORITY-DATA: 1998JP-0181503 (June 13, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2000002709 A	January 7, 2000		009	G01N033/68

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
JP2000002709A	June 13, 1998	1998JP-0181503	

INT-CL (IPC): C12N 15/02; C12P 21/08; G01N 33/53; G01N 33/68

ABSTRACTED-PUB-NO: JP2000002709A

BASIC-ABSTRACT:

NOVELTY - Diagnosis of leukemia comprising evaluating the presence of stanniocalcin in leucocytes using a specific antibody, is new. Stanniocalcin present in the blood cell fraction, excluding erythrocyte, is evaluated for diagnosing leukemia. DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for leukemia diagnosis kit comprising antibody specific for stanniocalcin protein and heat resistant DNA-polymerase.

USE - The method is useful for diagnosing leukemia.

ADVANTAGE - The diagnosis method is simple and specific.

ABSTRACTED-PUB-NO: JP2000002709A

EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/1

DERWENT-CLASS: B04 D16 S03

CPI-CODES: B04-B04D; B04-G01; B04-L04A; B11-C07A; B11-C08E3; B11-C08E5;

B12-K04A1; B12-K04F; D05-H09; D05-H11; D05-H14B;

EPI-CODES: S03-E14H; S03-E14H4;

L22 ANSWER 1 OF 11 BIOTECHDS COPYRIGHT 2002 DERWENT INFORMATION LTD

ACCESSION NUMBER: 2001-10582 BIOTECHDS

TITLE: Novel assay for diagnosing and monitoring cancer, involves determining levels of **Lng108** in cells, tissues or bodily fluids of the patient, and comparing with control; vaccine

AUTHOR: Recipon H; Macina R A; Chen S Y; Sun Y

PATENT ASSIGNEE: Diadexus

LOCATION: Santa Clara, CA, USA.

PATENT INFO: WO 2001032209 10 May 2001

APPLICATION INFO: WO 2000-US30482 3 Nov 2000

PRIORITY INFO: US 1999-163444 4 Nov 1999

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2001-316386 [33]

AN 2001-10582 BIOTECHDS

AB Diagnosing the presence of cancer or diagnosis of metastases of cancer in

a patient, involves determining levels of **Lng108** in a sample (S1) of cells, tissues, or bodily fluids in a patient and comparing the determined levels with levels of **Lng108** in sample (S2) of cells, tissues or body fluids from a normal human control. Also claimed are: staging cancer in a patient; monitoring cancer in a patient for the onset of metastasis; monitoring a change in stage of cancer in a patient;

identifying potential therapeutic agent for use in imaging and treating cancer; imaging cancer in a patient by administering an antibody specific

for **Lng108**; and a vaccine for treating cancer, by immunogenically stimulating the amount of **Lng108**. (36pp)

L28 ANSWER 1 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 97:267781 SCISEARCH
 THE GENUINE ARTICLE: WQ548
 TITLE: Mob-1, a Ras target gene, is overexpressed in colorectal cancer
 AUTHOR: Zhang R; Zhang H; Zhu W M; **Pardee A B**; Coffey R J; **Liang P (Reprint)**
 CORPORATE SOURCE: VANDERBILT UNIV, DEPT CELL BIOL, SCH MED, VANDERBILT CANC CTR, NASHVILLE, TN 37232 (Reprint); VANDERBILT UNIV, DEPT CELL BIOL, SCH MED, VANDERBILT CANC CTR, NASHVILLE, TN 37232; VANDERBILT UNIV, DEPT MED & CELL BIOL, NASHVILLE, TN 37232; VANDERBILT UNIV, VET AFFAIRS MED CTR, NASHVILLE, TN 37232; DANA FARBER CANC INST, DIV CELL GROWTH & REGULAT, BOSTON, MA 02115
 COUNTRY OF AUTHOR: USA
 SOURCE: ONCOGENE, (3 APR 1997) Vol. 14, No. 13, pp. 1607-1610.
 Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE, HAMPSHIRE, ENGLAND RG21 6XS.
 ISSN: 0950-9232.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: English
 REFERENCE COUNT: 17

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Mutations in the ras oncogenes have been linked to many different cancers, In contrast to the extensive body of knowledge related to the genetics of ras activation, relatively little is known of the transcriptional events triggered by ras, In previous work we have used differential display to identify Mob-1, a member of alpha-chemokine family, as one of the immediate transcriptional targets following Ras activation, Here, we provide additional experimental evidence to support this finding by the use of an inducible H-ras expression system, the treatment of Ras farnesyl transferase inhibitor and activation of endogenous Ras by serum growth factors, We further demonstrate that IP-10, the human homolog of Mob-1, is overexpressed in the majority of colorectal cancers.

L28 ANSWER 2 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 96:552791 SCISEARCH
 THE GENUINE ARTICLE: UY023
 TITLE: IDENTIFICATION OF MESSENGER-RNAS DIFFERENTIALLY EXPRESSED IN QUIESCENCE OR IN LATE G1 PHASE OF THE CELL-CYCLE IN HUMAN BREAST-CANCER CELLS BY USING THE DIFFERENTIAL DISPLAY METHOD
 AUTHOR: ALPAN R S; SPARVERO S; **PARDEE A B (Reprint)**
 CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST D810, DIV CELL GROWTH & REGULAT, 44 BINNEY ST, BOSTON, MA, 02115 (Reprint); HARVARD UNIV, SCH MED, DANA FARBER CANC INST D810, DIV CELL GROWTH & REGULAT, BOSTON, MA, 02115; HARVARD UNIV, SCH MED, DEPT BIOL CHEM & MOLEC PHARMACOL, BOSTON, MA, 02115
 COUNTRY OF AUTHOR: USA
 SOURCE: MOLECULAR MEDICINE, (JUL 1996) Vol. 2, No. 4,

pp. 469-478.
ISSN: 1076-1551.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 23

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background: The decision for a cell to enter the DNA synthesis (S) phase of the cell cycle or to arrest in quiescence is likely to be determined by genes expressed in the late G1 phase, at the restriction point. Loss of restriction point control is associated with malignant cellular transformation and cancer. For this reason, identifying genes that are differentially expressed in late G1 phase versus quiescence is important for understanding the molecular basis of normal and malignant growth.

Materials and Methods: The differential display (DD) method detects mRNA species that are different between sets of mammalian cells, allowing their recovery and cloning of the corresponding cDNAs. Using this technique, we compared mRNAs from synchronized human breast cancer cells (21PT) in quiescence and in late G1.

Results: Six mRNAs differentially expressed in late G1 or in quiescence were identified. One mRNA expressed 10 hr after serum induction showed 99% homology to a peptide transporter involved in antigen presentation of the class I major histocompatibility complex (TAP-1) mRNA. Another mRNA expressed specifically in quiescence and down-regulated 2 hr following serum induction showed 98% homology to human NADP(+)-dependent

cytoplasmic malic enzyme (EC1.1.1.40) mRNA, which is an important enzyme in fatty acid synthesis and lipogenesis. Three others showed high homology to different mRNAs in the GeneBank, corresponding to genes having unknown functions. Finally, one mRNA revealed no significant homology to known genes in the GeneBank.

Conclusions: We conclude that DD is an efficient and powerful method for the identification of growth-related genes which may have a role in cancer development.

L28 ANSWER 3 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 96:370736 SCISEARCH
THE GENUINE ARTICLE: UJ638
TITLE: BETTER GEL RESOLUTION AND LONGER CDNAS INCREASE THE PRECISION OF DIFFERENTIAL DISPLAY
AUTHOR: AVERBOUKH L (Reprint); DOUGLAS S A; ZHAO S; LOWE K; MAHER J; PARDEE A B
CORPORATE SOURCE: CHILDRENS HOSP, DANA FARBER CANC INST, DIV CELL GROWTH & REGULAT, 44 BINNEY ST, BOSTON, MA, 02115 (Reprint); SMITHKLINE BEECHAM PHARMACEUT, KING OF PRUSSIA, PA, 19406;
GENOMYX, FOSTER CITY, CA, 00000
COUNTRY OF AUTHOR: USA
SOURCE: BIOTECHNIQUES, (MAY 1996) Vol. 20, No. 5, pp. 918-921.
ISSN: 0736-6205.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 6

L28 ANSWER 4 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 95:799539 SCISEARCH
 THE GENUINE ARTICLE: BE08E
 TITLE: ANALYSIS OF ALTERED GENE-EXPRESSION BY DIFFERENTIAL
 DISPLAY
 AUTHOR: **LIANG P (Reprint)**; BAUER D; **AVERBOUKH L**
 ; WARTHOF P; ROHRWILD M; MULLER H; STRAUSS M; **PARDEE**
A B
 CORPORATE SOURCE: DANA FARBER CANC INST, DIV CELL GROWTH & REGULAT, BOSTON,
 MA, 02115 (Reprint); HUMBOLDT UNIV BERLIN, MAX DELBRUCK
 CTR MOLEC MED, MAX PLANCK SOC, D-13122 BERLIN, GERMANY;
 HARVARD UNIV, SCH MED, DEPT CELL BIOL, BOSTON, MA, 02115;
 DANISH CANC SOC, DIV CANC BIOL, DK-2100 COPENHAGEN,
 DENMARK
 COUNTRY OF AUTHOR: USA; GERMANY; DENMARK
 SOURCE: METHODS IN ENZYMOLOGY, (1995) Vol. 254, pp.
 304-321.
 ISSN: 0076-6879.
 DOCUMENT TYPE: General Review; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 22

L28 ANSWER 5 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 95:486111 SCISEARCH
 THE GENUINE ARTICLE: RH966
 TITLE: RNA GENETICS OF BREAST-CANCER - MASPIN AS PARADIGM
 AUTHOR: **SAGER R (Reprint)**; SHENG S; ANISOWICZ A;
 SOTIROPOULOU G; ZOU Z; STENMAN G; SWISSHELM K; CHEN Z;
 HENDRIX M J C; PEMBERTON P; RAFIDI K; RYAN K
 CORPORATE SOURCE: DANA FARBER CANC INST, BOSTON, MA, 02115 (Reprint);
 GOTHENBURG UNIV, SAHLGRENSKA HOSP, DEPT PATHOL, CANC
 GENET
 LAB, S-41345 GOTHENBURG, SWEDEN; UNIV WASHINGTON, DEPT
 PATHOL, SEATTLE, WA, 98195; ST LOUIS UNIV, CARDINAL
 GLENNON CHILDRENS HOSP, SCH MED, PEDIAT RES INST, ST
 LOUIS, MO, 63110; LXR BIOTECHNOL INC, RICHMOND, CA, 94804
 COUNTRY OF AUTHOR: USA; SWEDEN
 SOURCE: COLD SPRING HARBOR SYMPOSIA ON QUANTITATIVE BIOLOGY, (
 1994) Vol. 59, pp. 537-546.
 ISSN: 0091-7451.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 40

L28 ANSWER 6 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
 ACCESSION NUMBER: 95:343641 SCISEARCH
 THE GENUINE ARTICLE: QX634
 TITLE: RECENT ADVANCES IN DIFFERENTIAL DISPLAY
 AUTHOR: **LIANG P (Reprint)**; **PARDEE A B**
 CORPORATE SOURCE: DANA FARBER CANC INST, DIV CELL GROWTH & REGULAT, 44
 BINNEY ST, BOSTON, MA, 02115 (Reprint)
 COUNTRY OF AUTHOR: USA
 SOURCE: CURRENT OPINION IN IMMUNOLOGY, (APR 1995) Vol.
 7, No. 2, pp. 274-280.
 ISSN: 0952-7915.

DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Differential display and RNA arbitrary primed polymerase chain reaction are methods recently designed to identify and isolate differentially expressed genes. Methodological modifications have since been introduced to streamline the techniques. The major effort has centered on how to eliminate false positives as approached from a variety of angles, ranging from RNA sample preparation, northern blot confirmation, primer length variation, to better experimental design.

L28 ANSWER 7 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 94:405703 SCISEARCH

THE GENUINE ARTICLE: NV420

TITLE: CHRONIC CARDIAC REJECTION - IDENTIFICATION OF 5
UP-REGULATED GENES IN TRANSPLANTED HEARTS BY DIFFERENTIAL
MESSENGER-RNA DISPLAY

AUTHOR: UTANS U; LIANG P; WYNER L R; KARNOVSKY M J;
RUSSELL M E (Reprint)

CORPORATE SOURCE: HARVARD UNIV, SCH PUBL HLTH, CARDIOVASC BIOL LAB, 677
HUNTINGTON AVE, BOSTON, MA, 02115 (Reprint); HARVARD
UNIV,

SCH PUBL HLTH, CARDIOVASC BIOL LAB, BOSTON, MA, 02115;
HARVARD UNIV, SCH MED, BOSTON, MA, 02115; BRIGHAM &

WOMENS

HOSP, DIV CARDIOVASC, BOSTON, MA, 02115

COUNTRY OF AUTHOR: USA

SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
UNITED STATES OF AMERICA, (05 JUL 1994) Vol. 91,
No. 14, pp. 6463-6467.
ISSN: 0027-8424.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 21

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Transplant arteriosclerosis, the major manifestation of chronic rejection, develops after allogeneic (Lewis to F344) but not syngeneic (Lewis to Lewis) rat cardiac transplantation. To identify transcriptionally regulated mediators associated with chronic cardiac rejection, we adapted the differential mRNA display technique for in vivo transplant specimens. Gene transcript patterns in four allogeneic hearts showing early signs of chronic rejection were compared with those in two syngeneic hearts exposed to the same surgical procedure but

histologically

normal. Twelve differentially expressed cDNA bands were identified. We improved the probability of isolating one or more allograft-specific

cDNAs

from a single display band by first using recovered and reamplified PCR products as probes in RNA blot analysis. cDNA fragments cloned from individual bands were then used in a second RNA blot analysis, which allowed for the correlation of specific mRNA transcripts with cDNA

clones.

Five cDNA clones produced time dependent, allograft-specific hybridization. Sequence analysis demonstrated that two of these cDNAs corresponded to unknown genes, whereas the other three represented known

genes not previously associated with chronic rejection. The latter group included the macrophage lectin specific for galactose/N-acetylgalactosamine (a cell-surface receptor), the nuclear P1 gene (a homologue of a yeast replication protein), and a ubiquitin-like gene. Our application of the differential display technique allowed the direct identification of potential mediators under in vivo conditions that preserve the environment of the disease process-including infiltrating cell populations critical to the inflammatory response.

L28 ANSWER 8 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 94:367687 SCISEARCH
THE GENUINE ARTICLE: NP970
TITLE: A CAUTIONARY NOTE ON REACTION TUBES FOR DIFFERENTIAL
DISPLAY AND CDNA AMPLIFICATION IN THERMAL CYCLING
AUTHOR: CHEN Z H (Reprint); SWISSHELM K; **SAGER R**
CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST, DIV CANC
GENET, 44 BINNEY ST, BOSTON, MA, 02115 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: BIOTECHNIQUES, (JUN 1994) Vol. 16, No. 6, pp.
1002.
ISSN: 0736-6205.
DOCUMENT TYPE: Note; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 5

L28 ANSWER 9 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 94:50852 SCISEARCH
THE GENUINE ARTICLE: MT966
TITLE: MASPIN, A SERPIN WITH TUMOR-SUPPRESSING ACTIVITY IN HUMAN
MAMMARY EPITHELIAL-CELLS
AUTHOR: ZOU Z Q; ANISOWICZ A; HENDRIX M J C; THOR A; NEVEU M;
SHENG S J; RAFIDI K; SEFTOR E; **SAGER R (Reprint)**
CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST, DIV CANC
GENET, 44 BINNEY ST, BOSTON, MA, 02115 (Reprint); HARVARD
UNIV, SCH MED, DANA FARBER CANC INST, DIV CANC GENET, 44
BINNEY ST, BOSTON, MA, 02115; UNIV ARIZONA, CTR CANC,
TUCSON, AZ, 85724; MASSACHUSETTS GEN HOSP, DEPT PATHOL,
BOSTON, MA, 02114
COUNTRY OF AUTHOR: USA
SOURCE: SCIENCE, (28 JAN 1994) Vol. 263, No. 5146, pp.
526-529.
ISSN: 0036-8075.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: PHYS; LIFE; AGRI
LANGUAGE: ENGLISH
REFERENCE COUNT: 21

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A gene encoding a protein related to the serpin family of protease inhibitors was identified as a candidate tumor suppressor gene that may play a role in human breast cancer. The gene product, called maspin, is expressed in normal mammary epithelial cells but not in most mammary carcinoma cell lines. Transfection of MDA-MB-435 mammary carcinoma cells with the maspin gene did not alter the cells' growth properties in vitro, but reduced the cells' ability to induce tumors and metastasize in nude mice and to invade through a basement membrane matrix in vitro. Analysis of human breast cancer specimens revealed that loss of maspin expression occurred most frequently in advanced cancers. These results support the

hypothesis that maspin functions as a tumor suppressor.

L28 ANSWER 10 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 93:482472 SCISEARCH
THE GENUINE ARTICLE: LQ337
TITLE: IDENTIFICATION BY DIFFERENTIAL DISPLAY OF ALPHA-6
INTEGRIN
AS A CANDIDATE TUMOR-SUPPRESSOR GENE
AUTHOR: **SAGER R (Reprint);** ANISOWICZ A; NEVEU M;
LIANG P; SOTIROPOULOU G
CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST, DIV CANC
GENET, 44 BINNEY ST, BOSTON, MA, 02115 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: FASEB JOURNAL, (JUL 1993) Vol. 7, No. 10, pp.
964-970.
ISSN: 0892-6638.
DOCUMENT TYPE: Note; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 33

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A new method of differential expression cloning called differential display (DD) has been used to screen for novel tumor suppressor genes involved in breast cancer. The screen is based on positive selection at the mRNA level for genes expressed in normal mammary epithelial cells but decreased or lost in corresponding tumor cells. A candidate tumor suppressor gene recovered by DD is integrin alpha-6 (alpha6), a component of the heterodimeric integrin receptors alpha6beta1 and alpha6beta4. Loss of alpha6 expression was confirmed in total RNAs by Northern blot analysis and by immunostaining with alpha6 antibodies. Consistent with these cell culture findings, previous immunostaining of mammary tissue sections has identified decreased alpha6 protein expression during breast tumor progression. Southern blot analysis demonstrated that alpha6 gene is present in tumor cell lines, suggesting that re-expression may be inducible by pharmacological intervention. The likelihood that alpha6 may have tumor suppressing activity is supported by growing evidence of a central role for integrins in transducing growth control and differentiation signals from growth factors and the extracellular matrix (ECM).

L28 ANSWER 11 OF 11 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 93:461507 SCISEARCH
THE GENUINE ARTICLE: LN960
TITLE: DISTRIBUTION AND CLONING OF EUKARYOTIC MESSENGER-RNAS BY
MEANS OF DIFFERENTIAL DISPLAY - REFINEMENTS AND
OPTIMIZATION
AUTHOR: **LIANG P (Reprint);** AVERBOUKH L;
PARDEE A B
CORPORATE SOURCE: HARVARD UNIV, SCH MED, DANA FARBER CANC INST, DIV CELL
GROWTH & REGULAT, BOSTON, MA, 02115 (Reprint); HARVARD
UNIV, SCH MED, DEPT BIOL CHEM & MOLEC PHARMACOL, BOSTON,
MA, 02115
COUNTRY OF AUTHOR: USA
SOURCE: NUCLEIC ACIDS RESEARCH, (11 JUL 1993) Vol. 21,
No. 14, pp. 3269-3275.
ISSN: 0305-1048.
DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 16

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Differential display has been developed as a tool to detect and characterize altered gene expression in eukaryotic cells. The basic principle is to systematically amplify messenger RNAs and then distribute their 3' termini on a denaturing polyacrylamide gel. Here we provide methodological details and examine in depth the specificity, sensitivity and reproducibility of the method. We show that the number of anchored oligo-dT primers can be reduced from twelve to four that are degenerate

at the penultimate base from the 3' end. We also demonstrate that using optimized conditions described here, multiple RNA samples from related cells can be displayed simultaneously. Therefore process-specific rather than cell-specific genes could be more accurately identified. These results enable further streamlining of the technique and make it readily applicable to a broad spectrum of biological systems.

=>

L10 ANSWER 19 OF 19 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1996:403247 CAPLUS

DOCUMENT NUMBER: 125:83287

TITLE: Genetic changes during immortalization of human cells

AUTHOR(S): Reddel, Roger R.; Bryan, Tracy M.; Rogan, Eileen M.;
Noble, Jane R.; Maclean, Kenneth; Whitaker, Noel J.;
Chang, Andy C.-M.

CORPORATE SOURCE: Children's Medical Research Institute, Westmead,
2145,

Australia

SOURCE: Radiat. Oncol. Invest. (1996), Volume Date 1995-1996,
3(6), 299-306

CODEN: ROINEU; ISSN: 1065-7541

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Spontaneous immortalization of fibroblasts contg. an inherited mutation
of

one p53 allele was assocd. with loss of the wild-type p53 allele and loss
of p16INK4 gene expression, but this combination of genetic events was
insufficient for immortalization. Loss of p16INK4 expression appears to
be an alternative to loss of functional retinoblastoma gene product. In
all cell lines studied, immortalization was assocd. either with
stabilization of telomere length in the presence of telomerase activity

or

with the acquisition of long and heterogeneous telomeres in the absence
of

detectable telomerase. The role of other genes, such as human
stanniocalcin, in the immortalization process is under investigation.

L7 ANSWER 3 OF 9 MEDLINE DUPLICATE 2

ACCESSION NUMBER: 1998372535 MEDLINE

DOCUMENT NUMBER: 98372535 PubMed ID: 9708804

TITLE: High expression of **stanniocalcin** in differentiated brain neurons.

AUTHOR: Zhang K Z; Westberg J A; Paetau A; von Boguslawsky K; Lindsberg P; Erlander M; Guo H; Su J; Olsen H S; Andersson L C

CORPORATE SOURCE: Department of Pathology, Haartman Institute, University of Helsinki, Finland.

SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (1998 Aug) 153 (2) 439-45.
Journal code: 3RS; 0370502. ISSN: 0002-9440.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199808

ENTRY DATE: Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980825

AB **Stanniocalcin** (STC) is a glycoprotein hormone first found in fish, in which it regulates calcium homeostasis and protects against hypercalcemia. Human and mouse stc cDNA were recently cloned. We found a dramatically upregulated expression of STC during induced neural differentiation in a human neural crest-derived cell line, Paju. Immunohistochemical staining of sections from human and adult mouse brain revealed abundant presence of STC in the neurons with no activity in the glial cells. STC expression was not seen in immature brain neurons of fetal or newborn mice. Given that STC has been found to regulate calcium/phosphate metabolism in some mammalian epithelia, we suggest that STC may act as a regulator of calcium homeostasis in terminally differentiated brain neurons.

L7 ANSWER 4 OF 9 MEDLINE DUPLICATE 3

L16 ANSWER 5 OF 15

MEDLINE

DUPLICATE 3

ACCESSION NUMBER: 1999145369 MEDLINE
DOCUMENT NUMBER: 99145369 PubMed ID: 10022771
TITLE: Molecular cloning and characterization of
stanniocalcin-related protein.
AUTHOR: DiMattia G E; Varghese R; Wagner G F
CORPORATE SOURCE: Department of Oncology, The University of Western Ontario,
The London Regional Cancer Centre, Canada..
dimattia@julian.uwo.ca
SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1998 Nov 25)
146 (1-2) 137-40.
Journal code: 7500844. ISSN: 0303-7207.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990517
Last Updated on STN: 19990517
Entered Medline: 19990503

AB **Stanniocalcin** (STC) is a glycoprotein hormone first discovered
in fish and recently identified in humans and mice. In this report we
have described the cloning of an STC-like cDNA, designated here as STC
related protein (STCrP). Human STCrP (hSTCrP) cDNAs were isolated as
expressed sequence tags (ESTs) and predicted a polypeptide of 302 amino
acids, with 58%, homology to human STC (hSTC). Ten of the eleven 1/2
cysteine residues that in STC allow for dimerization of monomeric
subunits
were conserved in hSTCrP. By Northern analysis, three hSTCrP mRNAs were
detected and were most abundant in pancreas, spleen and kidney as well as
a variety of different transformed cell lines. The high degree of
sequence homology suggests that STC and STCrP may have been derived from
a
common ancestral gene.

L16 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:403247 CAPLUS

DOCUMENT NUMBER: 125:83287

TITLE: Genetic changes during immortalization of human cells

AUTHOR(S): Reddel, Roger R.; Bryan, Tracy M.; Rogan, Eileen M.;
Noble, Jane R.; Maclean, Kenneth; Whitaker, Noel J.;
Chang, Andy C.-M.

CORPORATE SOURCE: Children's Medical Research Institute, Westmead,
2145,

Australia

SOURCE: Radiation Oncology Investigations (1996),
Volume Date 1995-1996, 3(6), 299-306
CODEN: ROINEU; ISSN: 1065-7541

PUBLISHER: Wiley-Liss

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Spontaneous immortalization of fibroblasts contg. an inherited mutation
of

one p53 allele was assocd. with loss of the wild-type p53 allele and loss
of p16INK4 gene expression, but this combination of genetic events was
insufficient for immortalization. Loss of p16INK4 expression appears to
be an alternative to loss of functional retinoblastoma gene product. In
all cell lines studied, immortalization was assocd. either with
stabilization of telomere length in the presence of telomerase activity

or

with the acquisition of long and heterogeneous telomeres in the absence
of

detectable telomerase. The role of other genes, such as human
stanniocalcin, in the immortalization process is under
investigation.

L16 ANSWER 11 OF 15 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 ACCESSION NUMBER: 95:55329 SCISEARCH
 THE GENUINE ARTICLE: QA375
 TITLE: A HUMAN ORPHAN CALCITONIN RECEPTOR-LIKE STRUCTURE
 AUTHOR: FLUHMAN B; MUFF R; HUNZIKER W; FISCHER J A; BORN W
 (Reprint)
 CORPORATE SOURCE: UNIV ZURICH, DEPT ORTHOPAED SURG, CALCIUM METAB RES LAB,
 FORCHSTR 340, CH-8008 ZURICH, SWITZERLAND (Reprint); UNIV
 ZURICH, DEPT ORTHOPAED SURG, CALCIUM METAB RES LAB,
 CH-8008 ZURICH, SWITZERLAND; UNIV ZURICH, DEPT MED,
 CH-8008 ZURICH, SWITZERLAND; F HOFFMANN LA ROCHE & CO
 LTD,
 CH-4002 BASEL, SWITZERLAND
 COUNTRY OF AUTHOR: SWITZERLAND
 SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (
 05 JAN 1995) Vol. 206, No. 1, pp. 341-347.
 ISSN: 0006-291X.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 16

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A novel calcitonin receptor-like protein of 461 amino acids with seven
 putative transmembrane domains has been identified through molecular
 cloning in a cDNA library of the human cerebellum. 91% and 56% of the
 amino acids are identical in a rat orphan calcitonin receptor-like
 sequence and the human calcitonin receptor, respectively. 5.2 kb mRNA is
 predominantly expressed in the lung, heart and kidney. Specific binding
 of I-125-labeled salmon calcitonin and human calcitonin gene-related
 peptide-I to COS-7 cells transiently transfected with the receptor cDNA
 was less than 0.5%. Cellular cAMP accumulation was indistinguishable in
 cDNA transfected and non-transfected control COS-7 and renal tubular
 cells from the American opossum stimulated with human and salmon calcitonin,
 human calcitonin gene-related peptide-I and -II, human amylin, human
 adrenomedullin, lizard helodermin, salmon **stanniocalcin** and
 chicken parathyroid hormone-related protein. The receptor-like protein
 whose ligand remains to be discovered belongs to the family of receptors
 of calcitonin, parathyroid hormone, secretin, vasointestinal peptide and
 pituitary adenylate cyclase-activating polypeptide. (C) 1995 Academic
 Press, Inc.

L16 ANSWER 12 OF 15

MEDLINE

DUPLICATE 6

ACCESSION NUMBER: 92214238 MEDLINE
DOCUMENT NUMBER: 92214238 PubMed ID: 1666805
TITLE: Salmon **stanniocalcin** and bovine parathyroid
hormone have dissimilar actions on mammalian bone.
AUTHOR: Stern P H; Shankar G; Fargher R C; Copp D H; Milliken C E;
Sato K J; Goltzman D; Herrmann-Erlee M P
CORPORATE SOURCE: Department of Pharmacology, Northwestern University
Medical School, Chicago, IL 60611.
CONTRACT NUMBER: AR 11262 (NIAMS)
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (1991 Nov)
6 (11) 1153-9.
Journal code: 8610640. ISSN: 0884-0431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199205
ENTRY DATE: Entered STN: 19920515
Last Updated on STN: 19970203
Entered Medline: 19920504

AB **Stanniocalcin** (STC), a calcium-regulating glycoprotein hormone
isolated from the corpuscles of Stannius of salmon, was tested for
effects

on bone and calcium metabolism in mammalian species (rats and mice). STC
generally failed to alter serum calcium of parathyroidectomized rats at
concentrations equimolar with effective concentrations of parathyroid
hormone (PTH). STC did not increase cAMP in ROS 17/2.8 or UMR-108
osteosarcoma cells, OK kidney cells, fetal rat limb bones, or neonatal
mouse calvariae, and similarly failed to increase urinary cAMP in rats.
STC did not consistently stimulate resorption in any of the rodent bone
culture systems, although variable resorptive responses were elicited in
fetal mouse calvariae. The results indicate that this fish hormone has
limited, if any, PTH-like activity on calcium metabolism in mammalian
systems.

L16 ANSWER 13 OF 15 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 91:615462 SCISEARCH
THE GENUINE ARTICLE: GN465
TITLE: CONTROL OF CALCIUM REGULATING HORMONES IN THE VERTEBRATES
- PARATHYROID-HORMONE, CALCITONIN, PROLACTIN, AND
STANNIOCALCIN
AUTHOR: BONGA S E W (Reprint); PANG P K T
CORPORATE SOURCE: CATHOLIC UNIV NIJMEGEN, FAC SCI, DEPT ANIM PHYSIOL, 6525
ED NIJMEGEN, NETHERLANDS (Reprint); UNIV ALBERTA, SCH
MED,
DEPT PHYSIOL, EDMONTON T6G 2H7, ALBERTA, CANADA
COUNTRY OF AUTHOR: NETHERLANDS; CANADA
SOURCE: INTERNATIONAL REVIEW OF CYTOLOGY, (1991) Vol.
128, pp. 139-213.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: ENGLISH
REFERENCE COUNT: 334